LECTURES ON

ANGINA PECTORIS

AND ALLIED STATES

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TO

W. T. GAIRDNER, M. D., F. R. S.,

REGIUS PROFESSOR OF MEDICINE IN THE UNIVERSITY OF GLASGOW.

Dear Dr. Gairdner:

Please accept the dedication of this little volume in token of the appreciation which your cis-atlantic brethren feel of the value of your life and work in our profession.

Sincerely yours,

WILLIAM OSLER.

NOTE.

THESE lectures were delivered to the graduate class in medicine at the Johns Hopkins Hospital, and appeared in *The New York Medical Journal*, *1896*, vol. lxiv. They are here republished with slight additions and corrections.

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That I almost have caught a cardiacle.

The wordes of the Host to the Phisicien and the Pardoner.—CHAUCER.

LECTURES ON ANGINA PECTORIS AND ALLIED STATES.

LECTURE I.

History of the recognition of angina.—Heberden, Rougnon, Morgagni.—Literature.—Definition.—Forms of heart pain.—Classification of the forms of angina.—Physiology and pathology of the coronary arteries.

The history of the recognition of the disorder known as angina pectoris is connected with the names of three celebrated men—Heberden, Jenner, and John Hunter.

On July 21, 1768, Heberden read, at the Royal College of Physicians, a paper entitled Some Account of a Disorder of the Breast, which was published in vol. ii of the *Medical Transactions* of the College of Physicians, 1772. An extract from the original description must be quoted: "There is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it. . . . The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called angina pectoris.

"Those who are afflicted with it are seized while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away if it were to increase or to continue; the moment they stand still all this uneasiness vanishes. In all other respects the patients are at the beginning of this disorder perfectly well, and, in particu-

lar, have no shortness of breath, from which it is totally different."

Subsequently, in the celebrated *Commentaries* upon which our grandfathers in the profession were educated, Heberden gave a fuller account of his experience with the disease. The name which he adopted can not be regarded as altogether satisfactory, since it was already in use in designating affections of the throat, with which its literal meaning—a strangling—is much more in harmony. In one sense, however, the term is fairly appropriate, since, as noted by Gairdner, the words anxiety and anguish, expressive of two of the most prominent features of the disease, have a derivation from the same Greek word as angina.

In 1773, John Hunter had his first attack, which was graphically described by his nephew, Everard Home: "While he was walking about the room he cast his eyes on the looking-glass, and observed his countenance to be pale, his lips white, giving the appearance of a dead man. This alarmed him and led him to feel for his pulse, but he found none in either arm; the pain continued, and he found himself at times not breathing. Being afraid of death soon taking place if he did not breathe, he produced the voluntary act of breathing by working his lungs by the power of the will." In 1776 he had a second attack, and when convalescent he visited Bath. Here he was seen by his friend and pupil, Edward Jenner, of Berkeley; and one of the most interesting and sagacious letters of that distinguished man was written to Heberden, giving his diagnosis of John Hunter's case, and suggesting, for the first time, the probable association of disease of the coronary arteries with angina pectoris. The letter is worth quoting in full: * "When you are acquainted with my motives, I

* Baron's *Life of Jenner*, London, 1827.

presume you will pardon the liberty I take in addressing you. I am prompted to it from a knowledge of the mutual regard that subsists between you and my worthy friend Mr. Hunter. When I had the pleasure of seeing him at Bath last autumn I thought he was affected with many symptoms of the angina pectoris. The dissections (as far as I have seen) of those who have died of it throw but little light upon the subject. Though, in the course of my practice, I have seen many fall victims to this dreadful disease, yet I have only had two opportunities of an examination after death. In the first of these I found no material disease of the heart, except that the coronary artery appeared thickened.

"As no notice had been taken of such a circumstance by anybody who had written on the subject, I concluded that we must still seek for other causes as productive of the disease; but, about three weeks ago, Mr. Paytherus, a surgeon at Ross, in Herefordshire, desired me to examine with him the heart of a person who had died of the angina pectoris a few days before. Here we found the same appearance of the coronary arteries as in the former case. But what I had taken to be an ossification of the vessel itself, Mr. P. discovered to be a kind of firm, fleshy tube, formed within the vessel, with a considerable quantity of ossific matter dispersed irregularly through it. This tube did not appear to have any vascular connection with the coats of the artery, but seemed to lie merely in simple contact with it.

"As the heart, I believe, in every subject that has died of the angina pectoris, has been found extremely loaded with fat, and as these vessels lie quite concealed in that substance, is it possible this appearance may have been overlooked? The importance of the coronaries, and how much the heart must suffer from their not being able duly to perform their functions (we can not be surprised at the painful spasms), is a

subject I need not enlarge upon, therefore shall just remark that it is possible that all the symptoms may arise from this one circumstance.

"As I frequently write to Mr. H. I have been some time in hesitation respecting the propriety of communicating the matter to him, and should be exceedingly thankful to you, sir, for your advice upon the subject. Should it be admitted that this is the cause of the disease, I fear the medical world may seek in vain for a remedy, and I am fearful (if Mr. Hunter should admit this to be the cause of the disease) that it may deprive him of the hopes of a recovery."

In another letter * Jenner gives as his reasons for not publishing his views earlier an anxiety lest they should be a source of annoyance to his friend Hunter. "Soon after Mr. Paytherus met with a case. Previous to our examination of the body I offered him a wager that we should find the coronary arteries ossified. This, however, proved not to be exactly true; but the coats of the arteries were hard. . . . At this time my valued friend, Mr. John Hunter, began to have the symptoms of angina pectoris too strongly marked upon him; and this circumstance prevented any publication of my ideas on the subject, as it must have brought on an unpleasant conference between Mr. Hunter and me." He says that Mr. Cline and Mr. Home did not think much of his views. "When, however, Mr. Hunter died, Mr. Home very candidly wrote to me, immediately after the dissection, to tell me I was right."

The further details of Hunter's remarkable case are always referred to. From 1785, when he had a severe illness, the attacks became increasingly frequent, and were brought on particularly by exercise and by worry and anger; and, indeed, he was accustomed to say "that his life was in the hands of any

* Parry. *An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*, 1799.

rascal who chose to annoy and tease him." During the last few years of his life, though he did a large amount of work, the attacks seem to have been very frequent, and would come on after very slight exertion and while he was operating. As he had himself predicted, death came suddenly, in consequence of a fit of temper at a meeting of the governors of St. George's Hospital, October 16, 1793. When contradicted flatly, he left the board room in silent rage, and in the next room gave a deep groan and fell down dead. The coronary arteries were found to be converted into open bony tubes, and the aorta was dilated.

Attempts have been made by French writers to claim the priority in the description of the disease for Rougnon, professor of medicine in the University of Besançon. In a letter addressed to M. Lorry, dated February 23, 1768,* he describes the case and circumstances of the death of a Captain Charles. The patient had become asthmatic, and on walking fast had a sort of suffocation. Six weeks before his death he had complained to M. Rougnon of "*une gêne singulière sur toute le partie antérieure de la poitrine en forme de plastron.*" The attacks evidently occurred with great suddenness, and disappeared with equal abruptness. The chief stress is laid upon the feeling of suffocation, but it is evident that associated with it there was pain of great intensity; "*seulement une douleur gravative dans la région du cœur, lorsqu'il éprouvoit ses suffocations.*" Captain Charles died very suddenly, shortly after dining with his friends. The pericardium was fatty; the heart was large; there were no valvular defects; the coronary veins were enlarged "prodigiously"; no mention was made of the condition of the coronary arteries. Rougnon lays stress upon the obstruction in the lungs and excessive ossification of

* *Lettre à M. Lorry sur une maladie nouvelle.* Besançon, 1768.

the cartilages. He confesses, however, that the condition was very puzzling, and the autopsy not at all satisfactory to his friends: "*M. Charles est mort, disoient-ils, parce qu'il est mort.*"

I can not agree with Professor Gairdner, who says that "there was no trace of anything like a clinical description of angina pectoris in M. Rougnon's letter." * The suddenness of the attacks, the pain in the region of the heart, the abrupt termination, and the mode of death—during exertion after a full meal—favor the view that the case was one of true angina. (Note A.)

To Morgagni, not Rougnon, is due the credit of the first description of a single case. In the splendid section on aneurysm of the aorta, he describes angina pectoris accurately in Case V, referring to the paroxysms, the pain, the difficulty of breathing, the numbness of the left arm, and the effect of exertion. I read you here extracts from the case.

"A lady, forty-two years of age, who for a long time had been a valetudinarian, and within the same period, on using pretty quick exercise of body, she was subject to attacks of violent anguish in the upper part of the chest on the left side, accompanied with a difficulty of breathing and numbness of the left arm; but these paroxysms soon subsided when she ceased from exertion. In these circumstances, but with cheerfulness of mind, she undertook a journey from Venice, purposing to travel along the continent, when she was seized with a paroxysm, and died on the spot. I examined the body on the following day. . . . The aorta was considerably dilated at its curvature; and, in places through its whole tract, the inner surface was unequal and ossified. These appearances were propagated into the arteria innominata. The aortic valves were indurated." He remarks: "The delay of blood in the aorta, in the heart, in the pulmonary vessels, and in the vena cava, would occasion

* *Lancet*, 1891, i, p. 604.

the symptoms of which the woman complained during life; namely, the violent uneasiness, the difficulty of breathing, and the numbness of the arm."—(Cooke's *Morgagni.*)

There are those, indeed, who regard Seneca as the first to describe the affection, in the remarkable account which he gives of his own disorder. With this view Forbes and Gairdner agree, but Parry and Stokes do not. I quote from Parry the following translation of part of Seneca's graphic account: "The attack is very short and like a storm. It usually ends within an hour. I have undergone all bodily infirmities and dangers; but none appears to me more grievous. Why not? Because to have any other malady is only to be sick; to have this is to be dying." Seneca states, too, that his physicians called the disease a *meditatio mortis.*

The literature of angina pectoris has become very voluminous. English writers have contributed most largely to the clinical description of the disease. Perhaps the two most valuable articles are lectures xxxvii and xxxviii in Latham's *Clinical Medicine*, which you will find in vol. i of the New Sydenham Society edition of his works; and Professor Gairdner's essay in Reynolds' *System of Medicine.* The best recent expositions in French and German are to be found in Huchard's *Traité clinique des maladies du cœur*, second edition, 1893, which gives a most exhaustive account of the various forms of angina, and O. Rosenbach's *Die Krankheiten des Herzens*, 1896. I pass about for your inspection a number of the monographs and journal articles which I have collected on the subject. Parry's essay has become very scarce, but it is in all the larger medical libraries. Rougnon's *Lettre*, published at Besançon, 1768, is still more rare. The copy in the Surgeon-General's Library is the only one I have ever seen. I would ask you to look at the first part at least of these fly leaves, which I picked up in an old book shop a few years ago.

It is a letter to Dr. Heberden from a man who signs himself "Unknown," descriptive of his own case. He had seen, in the *Critical Review*, an extract from Heberden's original paper, and, recognizing his malady, he wrote in this letter one of the very best accounts which exists in the literature. It is particularly noticeable for two things: He clearly dissociated the pain of the attack from the *angor* or mental feature, and he first made use of the now hackneyed phrase describing the latter aspect as "an universal pause within me of the operations of Nature." Expecting a sudden death, he left orders that Heberden should examine his body. Within three weeks from the writing of the letter the dissection was made by John Hunter.

Definition.—In the consideration of a disease it is well, if possible, to start with a clear understanding, or at least some concise statement, of its nature, and of the characters of the manifestations by which it is recognized. With some disorders this is a very easy matter. For example, insufficiency of the aortic valves is a clearly defined affection, with, it is true, a diverse ætiology, a varied anatomical picture (from a trifling curl of the edges of a valve, to a clean shaving of a segment from the aortic ring); but with all its variations there are associated definite sequences and well-characterized signs.

Angina pectoris is not a disease, but a syndrome or symptom group (without constant ætiological or anatomical foundations) associated with complex conditions, organic or functional, of the heart and aorta. Pain about the heart of an agonizing character, occurring in paroxysms, is the dominant feature of all varieties of the syndrome. Used to define paroxysmal attacks of pain in the chest—breast-pang—we employ the term generically, qualifying the varieties by such names as true, false, hysterical, and voso-motor.

Before passing to the discussion of the varieties of angina pectoris let me refer briefly to the subject of

Heart Pain.—Disturbance of sensation is a most inconstant symptom of heart disease; the gravest affections are often painless; the most trifling may present the features of an intense neuralgia; while a very limited lesion may have as its sole manifestation paroxysms of agonizing pain.

The following abnormal cardiac sensations may be recognized:

1. Consciousness of the heart's action; a fluttering, a sense of goneness, the indefinable uneasiness associated with palpitation, a sense of tension in the chest with gasping, all or some of which are common phenomena in emotional states, in indigestion, neurasthenia, and hysteria.

2. Pain—darting, stabbing, tearing or boring, dull and heavy, or acute and piercing, steady or paroxysmal—varying in grades of intensity and in duration, often transient and trifling, as in dyspepsia and the tobacco habit, more enduring and severe in hysteria and neurasthenia, and occurring in paroxysms of an agonizing, intolerable character in the forms of angina. It often radiates over the area of distribution of certain of the cervical and dorsal nerves.

3. There is an element peculiar to certain conditions of the heart, often associated with, but which can not itself be properly characterized as pain—indeed, the patient often expressly states that it is not of the nature of physical pain—a sense of imminent dissolution, a mental anguish, which has been variously expressed by patients and writers as a pause in the operations of Nature, the very hand of death, *angor animi*, etc. This it is which constitutes the special feature in a majority of the cases of true angina.

Classification of the Forms of Angina Pectoris.—It may seem a refinement to subdivide and sort cases of a disorder

which is acknowledged to be only a symptom, or, as it has been expressed, a neurosal incident of cardio-vascular disease; but there are practical advantages which far outweigh any theoretical objections—advantages of the very greatest moment in prognosis and in treatment.

Following the work of Heberden, Parry, and others, there were cases reported as angina which did not belong properly to that category, and the disorder was confounded with cardiac asthma, which we now term cardiac dyspnœa. As early as 1812 J. Latham read a paper on certain symptoms usually but not always denoting angina pectoris (*Medical Transactions*, Royal College of Physicians). He remarks that when the extremities are cold, the countenance is bluish or purplish, the pulse is rapid, and respiration is performed with difficulty and in an upright position of the body, the practitioner has usually concluded that the disease is angina pectoris. The class of cases which he described were evidently orthopnœa and cardiac dyspnœa, associated chiefly with affections of the abdomen. He calls the state angina *notha*, spurious angina, the first time, so far as I am aware, that the term was used in literature.

Laennec recognized different degrees of intensity in angina, stating that it was "far from possessing the degree of severity attributed to it by many authors," and was evidently aware that it occurred commonly enough without indicating any serious disease of the heart or large vessels. "Angina pectoris, in a slight or middling degree, is extremely common, and exists very frequently in persons who have no organic affection of the heart or large vessels." *

By far the most important contribution to the recognition of varieties of angina pectoris was made by Walshe, who, in

* Forbes's edition of *Laennec*, fourth edition, p. 650.

his text-book on *Diseases of the Heart*, described a pseudo-angina pectoris, occurring particularly in women, and in the subjects of hysteria, spinal irritation, and various forms of neuralgia.

The recognition by Beau, Graves, Stokes, and others of the relation between the abuse of tobacco and attacks of angina led to the separation of the important group of toxic cases. Other forms of pseudo-angina which are described are those dependent upon reflex causes, and the vaso-motor type of Nothnagel.

In any long series, the cases of angina fall into two groups: those in which there are signs of lesion of the heart or arteries, or of both, and those in which all symptoms of organic disease are absent. This was the important division recognized by Forbes into organic and functional angina—the angina pectoris vera and the angina pectoris notha—the true and the pseudo-angina.

In looking over the cases which form the basis of these lectures, I find that they fall into the following groups: (1) Angina pectoris vera, and (2) angina pectoris notha, under which are grouped hysterical, vasomotor, and toxic forms.

The Coronary Arteries.—A few essential points in the anatomy and physiology of the heart may here engage our attention for a few minutes. The coronary arteries are the Abana and Pharpar of the vascular rivers, "lucid streams," which water the very citadel of life. By means of these injected specimens, which I pass around, you may refresh your memories on their distribution. The arteries are, as you see, large in proportion to the size of the organ to be nourished. From the position of their origin it is evident that they must be subject to blood pressure during both systole and diastole. The left coronary is usually the larger, and divides into two main branches: the circumflex or posterior, which runs in the groove between the left ventricle and auricle, and the anterior

or descending ramus. Note particularly the branches of the latter vessel, which runs in the anterior interventricular groove. You will see a very large branch, which is given off to the anterior wall of the left ventricle, and several branches which pass deeply into the septum. This anterior branch is the important one in the morbid anatomy of the coronary arteries, since it is by far the most frequently found the seat of extensive sclerosis or of embolism or thrombosis. It may be called the *artery of sudden death.*

From the date of Sir John Eric Erichsen's observations on the subject (1842) to the present the effects of closure of the coronary arteries have been much discussed. A very good historical summary is given by W. T. Porter in the *Journal of Physiology*, vol xv, 1893. It is remarkable how discordant are the statements of different observers. As this author remarks, seldom have the results of physiological studies been more at variance; there is no statement which is not denied, no fact which is not disputed. More recently Porter has again gone over the whole question with a great deal of skill, and I will give you here some of his conclusions.*

The frequency of the stoppage of the heart's action is in proportion to the size of the artery tied. Ligation of the smallest artery, the arteria septi, does not cause arrest; of the next in size, the coronaria dextra, fourteen per cent. of the ligations were followed by arrest; then comes the larger descendens with twenty-eight per cent.; and, finally, the circumflex, the largest artery of all, with sixty-four per cent.

The effect of closure of the coronary arteries on the blood pressure within the heart is of great importance. After the tying of a single vessel there is a diastolic rise of pressure, which is not compensated for by any increase of pressure in

* *Journal of Experimental Medicine*, vol. i, No. 1, 1896.

the coronary arteries; on the contrary, in them the pressure is falling, while that in the auricles is rising. It is known that the normal mean pressure in the auricles, and consequently in the coronary veins near their mouths, is very low. A rise of a few millimetres of auricular pressure might interrupt the entire coronary circulation. This is one of the most important points brought out by Porter's researches, and I quote here a paragraph on this point: "It must be acknowledged, then, that a rising auricular pressure after ligation may at length put a stop to the whole blood supply of the cardiac muscle, and, as this rise is often occasioned by the closure of a single vessel, it is plain that the entire coronary circulation can, in fact, be interrupted by the ligation of one coronary artery."

It has been much debated whether the coronary arteries are really terminal or end arteries. Anatomically, it may be shown that they are not, since an injection liquid can be made to pass from one artery through communicating branches into the other. All are agreed, however, that the anastomosis is not sufficient to permit collateral circulation to keep a vascular area alive after the distributing artery is blocked. The effect of plugging of the artery is the production of what is known as an anæmic infarct, a well-recognized pathological condition, the consideration of which need not detain us. A very important matter relates to the effect of plugging of the coronary arteries upon the heart-beat; the contractions become of the type known as fibrillary, and it is difficult or impossible to get the organ to resume the ordinary co-ordinated beats, though experimentally this has been done, even after fibrillary contraction has been established.

The relation of coronary-artery disease to angina pectoris, which was suggested by Jenner, has directed the very particular attention of writers to the changes in these vessels. It does

one good to look over the older literature, and to note the accuracy with which some of the cases have been recorded, particularly by Morgagni. Parry, too, gives an interesting series from the older writers. The subject is so extensive that I can not enter upon it here in great detail, but I may, perhaps, bring it before you with sufficient emphasis if I speak of the common sequences in connection with illustrative cases.

The coronary arteries are very subject to degenerative changes, particularly in persons who have passed the middle period of life. They may be affected alone or as part of a widespread disease of the vessels. For practical purposes we need not consider any other change than arterio-sclerosis in its various grades, from a trifling thickening to atheroma and rigid calcification. We must, however, recognize an affection of the orifices of the arteries, apart from the common degeneration of the trunks. A gradual narrowing of the orifice of a vessel may be quite as serious as extensive disease of the branches. There is a form of aortitis met with not infrequently in men between the ages of thirty and forty, who have had syphilis and who have worked hard and drank deep (*devotees* of Venus, Bacchus, and Vulcan), in which the intima is swollen, almost corrugated, with fresh translucent areas of endarteritis. I skip all considerations of its anatomy. Three serious sequences may follow: (*a*) Rupture of the aorta, sometimes only of the intima, as clean cut as with a razor, in half or a third of the circumference, sometimes with the formation of a dissecting aneurysm; (*b*) the slow development of the ordinary form of aneurysm of the arch; and (*c*) narrowing of the orifices of the coronary arteries. Angina attacks, sudden death, and slowly developing myocarditis and its sequences are the possibilities in this third category. I pass around this fine plate of Corrigan's, taken from the *Dublin Journal*, in which you see great swelling of the intima above the valves, due, as

Corrigan expressed it, "to an effusion of organized lymph between the lining membrane and the fibrous coat." The patient in this case, a man only thirty-nine years of age, suffered with severe attacks of angina.

Let me illustrate by these specimens some of the more common pathological conditions associated with disease of the branches of the artery. Here is an extraordinary heart, which illustrates how much of the coronary circulation can be cut off if the obstruction takes place gradually. The organ was taken from a man aged about thirty-six or thirty-seven, who had been an inmate for eighteen years of the Institution for Feeble-minded Children at Elwyn, Pa. He was a large, powerful imbecile, dumb but not deaf. He was very good tempered, did a great deal of work about the farm, and frequently did very heavy lifting. He never had epilepsy; he was not known to be short of breath, nor had he complained or indicated in any way that he was out of health. One afternoon he had a sort of fit, the face became very much congested, and he died in about half an hour. There was nothing special found in the brain. The heart, as you see, is large, and weighed twenty ounces. There was general hypertrophy with dilatation. There was quite extensive fibroid myocarditis, particularly in the anterior wall of the left ventricle, at the apex, and in the lower portion of the septum ventriculorum; the valves were normal. But what I wish you to examine most particularly is the state of the coronary arteries, which are freely dissected out. The left vessel is almost obliterated, only a pin-point channel remaining, while of the right artery the main division passing between the auricle and ventricle is converted into a fibroid cord!

It is much more common to find one artery extensively diseased, or even completely obliterated. Take, for example, this specimen, which was removed from a colored man, aged about

thirty-five, who had aortic insufficiency, with dyspnœa and œdema of the legs. He died suddenly, though he had for some weeks great dilatation of the heart and general anasarca. The aortic segments are curled and thickened; the ascending arch is greatly deformed, with a recent general endarteritis. There are a few calcareous plates. The right coronary artery is completely obliterated. There is no opening whatever on the aorta. The left vessel is dilated, and presents atheromatous patches. There are areas of fibrous myocarditis in the left ventricle, but in other respects the muscular substance of the heart does not look abnormal, and it is not fatty.

Here is a much more common condition. In this anterior coronary artery you see a firmly adherent thrombus, which completely occludes the descending branch, to the lumen of which it is firmly attached. It was taken from a man about fifty years of age, who had mitral-valve disease and had a good deal of cardiac dyspnœa. Early one morning he was seized with severe pain about the heart and shortness of breath, and died in a very few moments. Both coronary arteries were thickened and calcified, and presented atheromatous plates, but no doubt the sudden death was due to the blocking of the anterior branch of the left coronary artery by the thrombus.

When the occlusion has persisted for any length of time before death the condition of anæmic necrosis may be found. I am sorry not to have a fresh specimen to show you, but most of you have, no doubt, seen microscopic, if not macroscopic, examples. It is important in the dissection of the heart to slice carefully the septum and the wall, as these infarcts of the heart muscle are found in numbers directly proportionate to the care with which they are sought. We have not had any very large number of cases. They are much more common, I think, in hospitals with old chronic cases, or with which there are in connection large almshouses, as at the Blockley Hospi-

tal. I was much impressed at that institution with the number of cases of anæmic infarcts—many more than I saw at the Montreal General Hospital or have seen here. They occur most frequently in the walls of the left ventricle and in the septum, particularly toward the apex. When fresh they stand out beyond the level of the surrounding muscle, and are sometimes very firm, yellowish white, or even quite opaque white in color. With the fresh infarcts there may be old fibroid patches, into which ultimately these areas of anæmic necrosis are transformed.

To complete the series, I show you here sections of the descending branch of the left coronary artery, which you see is almost completely obliterated by an old, much-altered thrombus. This case illustrates another sequence of slowly developing coronary artery disease—namely, fibroid myocarditis at the apex, with weakening of the wall, and the gradual formation of aneurysm of the heart. The specimen was taken from the body of a large, powerfully built man whose heart symptoms developed with great abruptness, and who presented for many months an obscure train of symptoms pointing to serious disease of the myocardium.

Autopsies on cases of angina pectoris are not common. The man with a fresh thrombus in the anterior branch of the left coronary artery probably died in a paroxysm of angina, but he had not had previous typical attacks. As I will tell you later on, the affection is rare in hospital practice so that we do not have opportunities of making the inspection of the bodies of persons who have died of the disease.

And, lastly, a few words on the *innervation of the heart*, a cardinal point, inasmuch as the very essence of the angina paroxysm must rest on some profound disturbance in the function of the nerves. The newer methods of investigation have added considerably to our knowledge of the distribution of the in-

trinsic nerves of the organ. Doubtless some of you have seen in the pathological laboratory Dr. Berkeley's wonderful specimens illustrating the ultimate terminations of the filaments between and on the fibres.* In looking at them one realizes the truth of the remark of a recent author, that it is difficult to say in which the myocardium is richer, nerve elements or muscle fibres. Everywhere throughout the organ—in the tissues beneath the endocardium and pericardium, throughout the muscle substance, and about the blood-vessels—the nerves are in extraordinary profusion. The double nerve supply you know, from vagus and sympathetic, and the double function, the former controlling, checking, and inhibiting, the latter augmenting the force and hastening the frequency of the heart-beats. The researches of His junior and Romberg have shown that the ganglion cells of the heart, even those lying in the vagus branches, have the same origin as all other sympathetic cells. They differ in protoplasmic appearances and in other ways from the cells of the spinal ganglia. The rhythmic action of the heart is probably automatic, due to a power inherent in the muscular fibres, though this point is still in dispute. Of the functions of the nerves we know a good deal, of the functions of the ganglia nothing. His and Romberg suggest that from them are transmitted to the central nervous system infinitely delicately graded, unconscious impulses, which regulate the circulation reflexly through the vagus and accelerator. Of Kronecker's co-ordination centre our knowledge is still very indefinite—indeed, its existence has been called in question. I have seen Kronecker perform the experiment, and certainly when the point in the dog's heart is pricked—it is situated about the lower limit of the upper third of the ventricular septum—the organ becomes paralyzed in a state of fibrillary tremor, from which it does not recover. This point

* Described in *Johns Hopkins Hospital Reports*, vol. iv.

is within the area of distribution of the anterior coronary artery, the vessel oftenest found plugged by thrombus or embolus in cases of sudden death.

Do these cardiac nerves possess other properties? Have they also, with the special function, the endowment of receiving tactile and painful impressions? Certainly the heart is not an organ of very acute sensibility. The most extensive lesions, inflammatory, degenerative, and neoplastic, may not excite a single painful sensation. Pericarditis of the most intense grade, with deep involvement of the myocardium, may give not the slightest indication of its existence.

In experimental work, pinching of the heart muscle may excite reflex movements of the muscles of the body. There are a few interesting cases in the human subject in which the heart has been exposed by accident sufficiently to enable it to be grasped or touched. In the well-known case which Harvey gives * of the young Viscount de Montgomery, in whom Charles I was so much interested, in consequence of a fracture of the ribs on the left side, with excessive suppuration, the heart was exposed, and from Harvey's account was quite insensitive: "Nempe, in homine vivente et vegeto, citra ullam offensam, cor sese vibrans, ventriculosque ejus pulsantes videret, as manu tangeret. Factumque est, ut serenissimus Rex, una mecum, cor sensu tactus privatum esse agnosceret. Quippe adolescens, nos ipsum tangere (nisi visu, aut cutis exterioris sensatione) neutiquam intelligebat."

There is one other point of great importance. Sensory-nerve endings have been demonstrated in the arteries by Thoma, and recently Smirnow † professes to have demonstrated similar structures in the connective tissues of the heart, he thinks the sensory-nerve beginnings of the depressor nerve.

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* *Exercitationes de generatione animalium*, 1651, p. 311.

† *Anatomischer Anzeiger*, 1895.

LECTURE II.

ANGINA PECTORIS VERA.

ÆTIOLOGY. GENERAL DESCRIPTION OF THE DISEASE.

Incidence of the disease.—Station in life.—Sex.—Age.—Epidemic influences.—Heredity.—Gout.—Diabetes.—Syphilis.—Specific fevers.—Heart disease.—Locomotor ataxia.—General picture of the disease.

Incidence of the Disease.—As noted long ago by Sir Gilbert Blaine, angina pectoris is a rare affection in hospital practice. Gairdner criticises this statement rather sharply, and yet I think that a majority of hospital physicians would be found to support it. During the ten years in which I lived in Montreal, I did not see a case of the disease either in private practice or at the Montreal General Hospital. At Blockley (Philadelphia Hospital), too, it was an exceedingly rare affection. I do not remember to have had a case under my personal care. There were two cases in my service at the University Hospital. During the seven years in which the Johns Hopkins Hospital has been opened, with an unusually large "material" in diseases of the heart and arteries, and with many cases of heart pain of various sorts, there have been only four instances of angina pectoris. You will find the statement in Fagge's *Practice* (third edition, vol. ii, p. 26) that "the writer has never seen classical angina in hospital practice."

On the other hand, an individual consultant may see within a year more cases than occur in all the hospitals of his town

within the same period. In corroboration of this striking contrast between the incidence of angina pectoris in hospital and consulting work I may refer to the statistics of the Edinburgh Royal Infirmary, in which for the two years covered by the *Hospital Reports*, 1893 and 1894, there were five cases among a total of 8,868 medical cases. Compare with this the personal experience of the distinguished Edinburgh consultant, Dr. Balfour, who, in his recently issued work on *The Senile Heart*, gives an analysis of ninety-eight cases of angina pectoris seen within ten years. My individual experience embraces a series of sixty cases, forty of which may be regarded as true angina.

The predisposing causes of angina pectoris vera are those of arterio-sclerosis; that is to say, so intimately associated is the true paroxysm with sclerotic conditions of the coronary arteries that it is extremely rare apart from them. Men of muscular, even athletic build, who have been *devotees* of Bacchus and of Venus, form perhaps the largest contingent. Gout, syphilis, and hereditary influence the causation only so far as they tend to cause sclerotic changes in the arteries; but it would be altogether too narrow a view to suppose that the ætiology of the disease is identical with that of arterio-sclerosis. The one is so common and the other comparatively rare even among the individuals most prone to sclerosis, that there must be a third element, an indefinite something, which yet escapes our knowledge, but which is the essential factor in the production of this terrible affliction.

Station in Life.—As Sir John Forbes remarks, it is an attendant rather of ease and luxury than of temperance and labor; on which account, though occurring among the poor, it is more frequently met with among the rich, or in persons of easy circumstances. It is remarkable how many prominent individuals have succumbed to the disease. We may say of it

as Sydenham did of the gout, that more wise men than fools are its victims.

I do not know that any special occupation or profession predisposes to it, but the frequency with which physicians are attacked has been commented upon by several writers. In my list of sixty cases of all forms, there were thirteen medical men, eight of whom had true angina. This percentage is doubtless exceptional, and due, in part at least, to my nomadic habits, and wide acquaintance in the profession.

Sex.—From the earliest description of the disease, the remarkable preponderance of males who are attacked has been noted. Heberden says: "I have seen nearly one hundred people under this disorder, of which number there have been three women" (*Commentaries*). The statistics collected by Huchard give in two hundred and thirty-seven cases of true angina only forty-two in women. In my own series of forty cases of true angina there was only one woman.

Age.—The age at which it is most common is that of arterio-sclerosis—after the fiftieth year of life. Of the forty cases on my list there were only four under the fortieth year. One of these, a man, aged thirty years, had had syphilis five years before; the other case, a woman, aged thirty-two years, had mitral-valve disease; the third case had terrible attacks of angina following chronic pleurisy. In the fifth decade there were thirteen; in the sixth, thirteen; in the seventh, nine; and of one case I did not get the exact age. The average of the thirty-nine cases was about fifty-three years. Cases are reported in quite young individuals, even in children, but such are almost invariably the subject of chronic valvular disease or of adherent pericardium.

Epidemic, Imitative, and Emotional Influences.—Laennec was "of the opinion that the prevalent type of disease influences its development," and adds, "I have some years met

with it frequently, and hardly at all in others." You will find reference in the literature to so-called outbreaks of angina which have been reported by Kleefeld * and by Gelineau.† I can not see that the cases recorded by Kleefeld have anything to do with angina pectoris. He describes the epidemic as a remittent fever with gastric complications, and much pain about the heart. Some of the cases were fatal, but no autopsies were made. Young persons, chiefly women and children, were attacked.

Gelineau, surgeon to the French corvette L'Embuscade, reports a remarkable outbreak among the sailors during a prolonged cruise in the Pacific. Scurvy had broken out and the men were much debilitated and anæmic. They became subject also to a severe dry colic. Following this there were many cases of angina. The first case was that of an old sailor, scorbutic and anæmic, who while climbing the mast was seized with intense pain about the heart. Five days after, five other men were attacked in the same sudden way, and three days later, three more. Gelineau lays a good deal of stress upon tobacco as a factor in the causation of the pain, and also upon the debility following the scurvy, dysentery, and dry colic. The effect of imitation, that extraordinary occult influence so potent in many forms of hysteria, must, no doubt, be taken into account. Perhaps the most notable instance is given by Dr. Taber Johnson in his report of Mr. Sumner's case.* "I have observed a curious fact, which it may be interesting to refer to here. I mean the unusual number of patients suffering from this disease, who, previous to Mr. Sumner's severe illness, had never supposed that they had any disease of the heart. This fact has been referred to by newspaper corre-

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* *Journal d. pract. Heilkunde*, 1823, lvii.

† *Gazette des Hôpitaux*, 1862, xxxv.

‡ *Boston Medical and Surgical Journal*, 1874.

spondents—viz., that during the illness of Mr. Sumner, and especially since his death, instances of its occurrence have considerably increased, and especially among those who strongly sympathized with the late senator. This seemingly sympathetic cause of disease has been noticed in other cases. I have been consulted by as many as thirty individuals, since Mr. Sumner's death, who imagined they were afflicted with his complaint. In some of these cases there was organic disease of the heart, but in a majority of them there was no cardiac trouble at all. Two weeks after the autopsy in Mr. Sumner's case, one of the physicians who assisted, a devotedly attached friend of the deceased, died of angina pectoris. I am informed that Dr. Hitchcock had but a few attacks, and that, prior to Mr. Sumner's death, he had never been a sufferer from angina pectoris."

Dr. Johnson says that he himself suffered from two attacks very closely resembling, if they were not really, angina. One of these occurred immediately after Mr. Sumner's death, and Brown-Séquard, who was present, said the phenomena were undoubtedly those of a paroxysm of angina. Twenty-two years have passed, and, happily for himself, as well as for our brethren of the District of Columbia, Dr. Taber Johnson has now less mobile nerves.

In Case X of my series of pseudo-angina the patient's husband died suddenly in a paroxysm of true angina.

Mental worry, severe grief, or a sudden shock may precede directly the onset of the attacks. In Case XXXVI, the paroxysms came on after the shock of the announcement that a son had committed suicide.

Heredity.—True angina pectoris is an arterial incident, and since the members of certain families show a special tendency to arterial degeneration, it is not surprising to find cases in father and son, or in brothers, or even in representatives of

three generations. There are remarkable instances on record. The first, and one of the most remarkable, is that reported by Dr. Robert Hamilton,* in which the father of the patient, a young man aged twenty-four, two brothers, and one sister were affected. In all, the disease developed in early life; in Hamilton's own patient, at the twelfth year. It is quite possible from his description that the disease may not have been angina pectoris, but spasmodic asthma associated with heart pain.

The best-known instance is that of the Arnold family. William Arnold, collector of customs of Cowes, died suddenly of spasm of the heart in 1801. His son, the celebrated Thomas Arnold, of Rugby, whose case I will narrate to you shortly, died in his first attack. Matthew Arnold, his distinguished son, was a victim of the disease for several years, and died suddenly in an attack on Sunday, April 15, 1888, having been spared, as he hopes in his little poem called A Wish—

> "the whispering, crowded room,
> The friends who come, and gape, and go;
> The ceremonious air of gloom—
> All, which makes death a hideous show!"

At the time of his death, the accounts which appeared in the *Lancet* and *British Medical Journal* were not clear as to the existence of attacks of angina. The various stages in the progress of his illness can be traced very well in his *Letters*,† in which you will find an account of numerous attacks from May, 1885, until the time of his death. (Note B.)

In looking over the literature one finds occasional references to cases occurring in several members of one family.

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* *Medical Commentaries*, 1785, ix.

† *Letters of Matthew Arnold.* Macmillan & Co., 1896.

Cazanave de la Roche * records three cases in one family—a sister, who was affected at the time of the report, and two brothers who had died of the disease. In Case XXIII on my list the patient's father died of angina pectoris.

Gout.—The relation of certain constitutional disorders to angina pectoris has been much discussed. The importance of gout as a factor was early suggested, and in this interesting little monograph of Butter's, which I show you here—the first separate treatise on the disease—the author places the seat of the disorder in the diaphragm, and calls it diaphragmatic gout. The affection has also been termed asthma arthriticum.

Nathaniel Chapman advocated strongly the arthritic nature of angina pectoris, and there can be no question, I think, that in a certain number of the victims gout plays an important rôle in inducing the arterio-sclerosis.

I have been particularly interested in examining into this point in the cases which have come under my observation within the past four or five years. There are four cases at least of my series in which gout seemed to play a part. Dr. ——, of Virginia, seen April 3, 1894, a very robust, vigorous man of forty-eight, temperate, a hard worker, who had not had syphilis, and in whom the attacks were fairly characteristic, thinks that gout (which is in his family) is directly responsible for the attacks. Certainly, after using without benefit for many months the iodides and the nitrites, he obtained the greatest relief from a prolonged course of colchicum. It is now more than two years since I saw him, and he remains well. In another case, a patient with attacks of angina pectoris *sine dolore*, there had been attacks of acute articular gout. In a third case, a man aged sixty-four, the upper half of the pinna of the lobe of the right ear was firm and calcified,

* *La Tribune médicale*, 1895, p. 832.

and the same process was beginning in the left ear. There were no tophi, but the calcification was, to say the least, suggestive. A fourth case was that of a physician from North Carolina, aged forty-six, who had for many years attacks of gouty arthritis, chiefly in the big toe, less frequently in the ankles. There was a well-marked tophus in the right ear.

Diabetes.—The association of angina pectoris with diabetes has been frequently noted. No instance has fallen under my personal observation. You will find the whole subject very thoroughly discussed by Ebstein in a recent paper in the *Berliner klinische Wochenschrift* of last year (1895).

Syphilis is one of the potent factors in inducing arteriosclerosis, and thus indirectly plays a rôle in angina pectoris. Of the cases in my series, only four gave a history of syphilis. The instances of aortitis to which I have already referred, occurring in the third and fourth decades in men who have had syphilis, have worked hard, and have been heavy drinkers, are sometimes associated with severe attacks of angina. In Case I, Lieutenant X., aged thirty years, a robust, powerful man, had had syphilis six years before his visit to me. The secondary symptoms were slight, and he had not had very thorough treatment. A year before I saw him he began to have severe pains in the heart, recurring in paroxysms, and associated with pain down the left arm, and dyspnœa on exertion. There was no perceptible enlargement of the heart; there was a systolic murmur at the apex and a soft bruit at the aortic area, without special accentuation of the aortic second sound. The attacks had been of such severity that he had been off duty for many months. He improved very much upon the iodide of potassium, but still had attacks six months after I saw him, since which time I have not heard of him. Corrigan's case, you remember, the illustrative plate of which I showed you at the last lecture, was in a young man, and belonged to this

group. The frontispiece in Balfour's work on the heart (second edition) illustrates another case of the same kind in a still younger man, aged twenty-four years. The angina attacks were associated with an aortitis which narrowed greatly the orifices of the coronary arteries.

Specific Fevers.—In connection with the specific fevers several writers have described angina-like attacks. Fraentzel, in his *Vorlesungen über die Krankheiten des Herzens* (Berlin, 1889), describes attacks of angina pectoris in the weakened and dilated heart following the infections, particularly erysipelas, typhoid fever, and pneumonia. J. W. Moore * has reported two instances of angina symptoms in connection with heart weakness during and after the specific fevers. In the epidemic of a remittent fever reported by Kleefeld (and already referred to) the attacks of heart pain may have been of this character. I do not remember to have seen a case in which the attack developed during convalescence from one of the ordinary fevers.

Among the many nervous sequelæ of *influenza*, few are more distressing than the attacks of severe cardiac pain. In some cases, indeed, the disease seems to have been the starting point of attacks of true angina. The frequency of the complication in the practices of some physicians is remarkable. In a paper on The Action of Influenza Poison on the Heart, Curtin and Watson state that within two years they met with fully seventy cases of painful attacks about the heart. The illustrative cases in their paper † show that some of the attacks must have been of very great severity, but, in most instances, the duration of the disease was short and the cases evidently belonged to the category of pseudo-angina. I have seen but

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* *Dublin Medical Journal*, 1890, vol. lxxxix.

† *International Medical Magazine*, January, 1893.

two instances in which the attack seemed to follow directly upon the influenza. One is certainly pseudo-angina; the other proved to be the genuine disease.

I saw on several occasions in Toronto a medical friend who, after a tolerably severe attack of influenza about three years ago, began to have attacks of agonizing pain about the heart. They came on without warning, the pain appearing in various parts of the chest, commonly under both shoulder blades, and especially severe in both wrists. There was at first no irregularity of the pulse or difficulty in breathing; but in some attacks there were piping râles during expiration. At first these attacks were almost nightly; several times they ended in vomiting (preceded by profuse salivation), the passage of more or less flatus, and copious sweating. There was no mental anxiety whatever, except, as he expressed it, "the pain was so intense that I was afraid I would recover, in order to endure it again." The pain in the arms was chiefly in the front of the wrists. The patient had not had any serious illness previously, had never had syphilis, had not been a heavy drinker, but had been a pretty heavy smoker. The attacks recurred with intensity throughout the early part of January. When I saw him there were no signs of cardiac disease. He had had a good deal of digestive disturbance. During the following summer and autumn he progressively improved, and I heard from him recently to the effect that now only in any extra strain, an in the attendance upon a difficult case of labor, does he feel any pain. He used the iodide steadily for some time without any special benefit. He attributes more benefit to lavage of the stomach with hot water night and morning. How far the influenza in this case was responsible for the attack is, of course, difficult to say, but when I saw him first he was very insistent that it was the cause of his whole trouble. From the rapid way in which the attacks have ameliorated and his present general condition there is, to say the least, a strong probability that it is functional and not associated with organic disease.

The other case was that of the late chief justice of this State, who had, in the early winter of 1893, a very severe attack of

influenza with much fever and prostration. In the latter part of December he began to have pain about the heart in walking briskly up a hill. Then he had more severe attacks, but in the summer of 1894 he was better, and was able to take long walks. The attacks recurred about Christmas, 1894. I saw him on January 20, 1895. There was no enlargement of the heart, the sounds were clear, the second aortic a little accentuated. The only striking anomaly was a condition of trigeminal heart-beats—groups of three beats, with an interval, followed in regular sequence. He improved very much through the summer of 1895. In October he had a severe shock on hearing of the sudden death from angina of his brother-in-law (Case XXXV on my list). He did not, however, have any recurrence until December. I saw him on January 5, 1896. The paroxysms had become more frequent and very severe. In the following week he died in an unusually prolonged attack. The onset of the angina corresponded with the period of convalescence from the influenza, which he always insisted had caused the attacks.

Heart Disease.—Paroxysms of agonizing substernal pain, with radiation to the neck and arm, are rare in the ordinary forms of heart disease which we meet with in hospital work. Heart pain is common enough, and if we counted all such cases as angina we would not have to lay stress on the infrequence of this syndrome in the wards. You remember the small boy in Ward F during the early part of this session, with greatly enlarged heart, probably from pericardial adhesions. Pain was the most distressing symptom of the case, but it had neither the intensity, the paroxysmal character, nor the accompaniments which warrant the diagnosis of true angina. So, too, in the case of the old colored woman, at present in Ward O, with mitral-valve disease and extreme arterio-sclerosis. I have pointed out to you that the attacks of sudden breathlessness and distress with transient pain, are of the nature of cardiac asthma, with which, as I will tell you later, angina pectoris is often confounded. Then, again, you have

to bear in mind the common complaint of pain beneath the left breast in patients with chlorosis and various forms of anæmia.

Of valvular affections, aortic insufficiency is that with which angina pectoris is most frequently associated. Of the forty cases in my list three presented signs of this lesion. The subjects of the degenerative type of the disease, which develops in men after the fortieth year, are much more prone to angina than those in whom the insufficiency has followed endocarditis. The younger the subject, the greater the probability that the incompetency results from an acute aortitis, as in Corrigan's case, to which I have referred on several occasions.

Angina pectoris is excessively rare in mitral-valve disease. This is well illustrated by Nothnagel's experience.* Of fifteen hundred cases of valvular disease of the heart seen in hospital and private practice, very many of which had symptoms of angina, there was but a single case in which the syndrome occurred in connection with mitral stenosis. Only one of my cases, a woman, had a mitral lesion. By far the most common heart disease with which angina is associated is chronic myocarditis, the signs of which are often dubious.

Cases of adherent pericardium and of aneurysm of the aortic arch may present the features of typical angina, more often, in my experience, of constant substernal pain or of cervico-brachial neuralgia.

A majority of the subjects of angina present the signs of arterio-sclerosis, with accentuation of the aortic second sound and slight increase in the area of transverse heart dullness. Some of the most rapidly fatal cases are those in which the physical signs are very slight, or even absent. Of the cases on

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* *Verhandlungen des Congresses f. innere Medicin*, Bd. x.

my list, in four only was the physical examination negative; three presented apical or basic murmurs; of the remainder, all of whom showed signs of sclerosis of the arteries, nine had indications of myocardial changes.

Locomotor Ataxia.—Considering the close relationship of syphilis to this disease, in which also arterio-sclerosis is so common, it is not surprising that attacks of angina pectoris should occur. No instance has fallen under my personal observation. You know that aortic insufficiency is not rare in tabes. At Blockley the association was a matter of every-day comment, and in the physical-diagnosis class we would send to the out wards for the old tabetics to demonstrate the lesions of arterio-sclerosis, and if not of aortic incompetency, of the ringing metallic aortic second sound, which so often accompanied the dilated and rigid aortic arch. You will find the subject fully discussed by Leyden in the *Zeitschrift f. klin. Medicin* for 1887, and since his paper there have been several less important communications.

General Picture of the Disease.—In any long series of cases of angina we can recognize four groups:

I. *Sudden Death, without other Manifestations of Angina Pectoris.*—Much more true of angina pectoris is what Andral said of the fulminant form of cholera: it begins where other diseases end—in death. The affection has indeed been called by Sir Walter Foster a mode of death, which reminds one of the expression of the physicians who spoke of Seneca's malady as a *meditatio mortis.* No inconsiderable proportion of sudden deaths in men of middle age and robust habits result from coronary-artery disease, from the rapid culmination, so to speak, of a condition which, in another (or on previous occasions in the individual himself), would have caused an ordinary attack of angina. Before all is over there may be a momentary conscious agony expressed by a cry, but in other in-

stances (and this is most frequently the case in the subjects of angina) the death is literally instantaneous; more rapid, perhaps, than that which occurs by any other mode.

Of the fifteen deaths in my series, eight took place suddenly; in five, gradually by cardiac asystole; in one, I did not learn the exact mode of death; in another, the patient died of obstruction of the bowels. Of the eight cases, in five death was sudden, almost without warning, and not in a paroxysm of angina.

Mr. S. (Case XXVI) died on his doorstep; Mr. W. (Case XXVII) died as he was leaving a friend's house; Dr. X. (Case VIII) died as he was walking from one room to another. He had had cardiac arrhythmia, Cheyne-Stokes breathing, and marked mental disturbance; Mr. E. (Case XXXV) died instantly on the edge of the bed as he was recovering from his first attack of angina, not having had pains for nearly twenty-four hours; Mr. R. (Case XI) fell over dead on attempting to get out of bed. The literature abounds in cases of this sort, and the proportion of the victims of angina who die abruptly is much larger than my figures indicate. Forbes mentions that of sixty-four cases sudden death occurred in forty-nine. Anatomically it has been shown that lesion of the coronary arteries is almost invariably present—either extensive arteriosclerosis, embolism, thrombosis, or in rare instances the bursting of a small atheromatous abscess in one vessel, such as killed the celebrated sculptor Thorwaldsen.* An explanation of the awful suddenness—"Life struck sharp on Death"—is probably to be found in the arrest of the heart in fibrillary contraction, such as takes place experimentally in animals after ligation of a coronary vessel.

II. *Death in the First Well-marked Paroxysm.*—A man

* Virchow's *Archiv*, Bd. xxv.

in full health, in the prime of life, may be seized with a paroxysm of angina, and die within a few hours. The cases in this category are not numerous. Perhaps the most remarkable one on record, which has become quite historic, is that of the celebrated Dr. Arnold, of Rugby, who in the words of his distinguished son (also a victim of the disease), arose

> "... to tread
> In the summer morning, the road
> Of death, at a call unforeseen,
> Sudden."

The following is Latham's account: *

"T. A. was within a day of completing his forty-seventh year. Up to a few hours before his death, both body and mind seemed equally to give proof and promise of health. He still took his accustomed pleasure and refreshment in strenuous exercise. His thoughts were still busily employed upon the highest subjects, conceiving and composing with wonderful ease, rapidity, and power. He retired to rest at midnight on the 11th of June, 1842, feeling and believing himself to be in perfect health. At a quarter before seven the next morning his medical attendant was called. What had previously occurred and what followed I will give in the words of Dr. Bucknill, who was with him during the short remaining period of his existence. 'On my entering his room he said that he was sorry to disturb me so soon; and that he had not sent for me before, thinking that it would go off. He added, "I have had very severe pain in the chest since five o'clock, at intervals, and it gets worse, I think." This pain was seated at the upper part of the chest, toward the left side, and extended down the left arm. He had been rather sick. He then asked me what the pain was. "What is it?" He was now almost free from pain. His pulse I could scarcely feel. The tongue was clean. There was cold perspiration over his face. The feet and legs were

* Latham's *Works*, vol. i, p. 453; New Sydenham Society, 1876. See also Stanley's *Life of Thomas Arnold.*

cool. The breathing at this time not troubled. I gave him immediately some hot, strong brandy and water, and having ordered a mustard plaster for his chest, till this was ready I applied hot flannels, and had his legs and arms rubbed and the feet wrapped up in flannels wrung out of hot water and mustard. The pulse became natural, the extremities more warm, and he was free from pain. The mustard plaster was brought and put on. It was not large enough and I ordered another. The pain then returning, I gave him more brandy and water, and it soon left him. And now he asked me again what the pain was. I told him I believed it was spasm of the heart. He exclaimed, "Ah!" I asked him whether he had ever fainted in his life. "No, never." If he had at any time difficulty of breathing. "No, never." If any pain in his chest before. "No, never." I then asked him if any of his family had ever had any disease of the chest. "Yes, my father had; he died of it." He inquired if disease of the heart was suddenly fatal. I answered that it was. "Was it a common disease?" I said not very common. "Where do you find it most?" "In large towns, I think." "Why?" "Perhaps from anxiety and eager competition among the higher, and intemperance among the lower classes." He was then quiet and free from pain, and I proposed to leave him for a minute or two. He had no pain whatever in my absence. On my return the perspiration was still in drops upon his forehead. The pulse was again feeble, and I gave him more brandy and water and had the flannels with mustard renewed. An attack of pain was coming on. He said, "I must stretch myself." I took one of his hands and held it until the pain was gone off. It was of short duration. I said, "Is it gone?" He answered, "Yes, entirely," adding that he "could scarcely bear it if it were as severe as it had been." He then asked me "what was the general cause of this kind of disease." He then said, "Is this likely to return?" I answered that I was afraid it was, but that, as the attacks had been less severe and less frequent, I hoped they would pass off. He next asked me if the disease was generally suddenly fatal. I said generally (for those who knew him were aware that it was impossible not to tell him the exact truth). I then asked him if he had any

pain. He said, "None but from the blister; one can bear outward pain, but it is not so easy to bear inward pain." I was now dropping some laudanum into a wineglass, when he inquired what I was going to give him. I told him laudanum, Hoffman's anodyne, and camphor; and, while I was preparing the mixture, and before I had finished, I heard a rattling in the throat and a convulsive struggle. I called out, and on turning to him I supported his head, which was thrown back on my shoulder. His eyes were fixed and his teeth set, and he was insensible. His breathing was very laborious, his chest heaved, and there was a severe struggle over the upper part of the body. His pulse was imperceptible, and after deep breathings at a few prolonged intervals all was over. He died in little more than half an hour after I first saw him.'"

The examination showed a soft, flaccid heart muscle. There was but one coronary artery, and that, considering the size of the heart, of small dimensions. It presented also a slight atheromatous deposit an inch from its orifice.

In no case in my series did death occur in the first paroxysm. The most rapid case was Mr. E. (Case XXXV), who had an agonizing paroxysm at 2.30 P. M. on October 14th, and several lesser recurrences throughout the night. There was no attack on the 15th, and he passed a comfortable night. On the 16th, at 9.10 A. M., he sat up on the edge of the bed to be helped to the commode, and fell over dead, about forty-two hours from the onset of the first attack.

III. *Recurring Attacks extending over a Period of Months or Years.*—Much more commonly a victim of angina pectoris has many paroxysms over a period of many months, or from three or four to twenty or even twenty-five years. The recurrences may be at long intervals, as in John Hunter's case, or they may render the patient's life unbearable, since he feels that the slightest transgression, muscular or emotional, may precipitate a paroxysm. Many a poor sufferer has felt what Senator Sumner expressed: "This treacherous disease pro-

duces in my mind a positive uncertainty, when I go out of my house, whether I shall ever enter it again a living man, and, with the pain I have to suffer, makes my life such a burden that the sooner it does its work the better I shall be pleased. Life, at the price I have to pay, is not worth the having." Let me read you the history of a typical case of this sort:

Case XXVI.—Mr. S., an editor by occupation, aged fifty-five, consulted me January 16, 1894, complaining of attacks of agonizing pain in the region of the heart. The patient was of a nervous temperament, but had been a very healthy man. He had never done hard physical work and had been moderate in the use of alcohol and tobacco. He did not think that he had ever had syphilis. Three years ago, following upon the shock of the announcement of the suicide of a son, he had his first attack of severe pain about the heart. Ever since, the attacks have recurred at irregular intervals, at first of a few weeks or a month, but within the past year they have been very frequent, so that he now rarely passes a day without paroxysms. They vary a great deal in intensity. If he walks fast or makes any unusual exertion he is stopped by an intense pain in the heart, and he has to pant for breath. After lasting for half a minute or so the pain passes off, and he is able to resume his walk. Any unusual emotion or excitement will bring on an attack at once. He not uncommonly now has as many as a dozen or more attacks in the day. In the severer paroxysms he feels as if the throat was greatly swollen, and says that both his throat and his temples throb, and that he gets very red in the face. As the attacks pass off he usually sweats quite profusely. From what I can gather, he did not appear to have had paroxysms of terrible agony, in which the sense of impending death was present. He says, however, that the feeling is as though the heart was grasped in a vice, and the pains shoot up the neck and down the left arm. Two weeks ago, in Philadelphia, while walking to the station, he felt an excessively severe pain in the chest, became short of breath, and fell unconscious. When he recovered he found himself in a neighboring chemist's

shop. He was able, however, to proceed on his journey. While in my waiting room this patient had two attacks, and while I was examining him he had a third, the phenomena of which I will describe to you later. Three days after his visit to me, while walking up the steps of his house, he dropped dead.

The great majority of all cases of angina pectoris come in this group.

IV. *Rapidly Repeated Attacks over a Period of Days or Weeks, with the Development of a State of Cardiac Asystole —l'état de mal angineux.*—An individual in apparently good health, who may not have had any indications of heart trouble, or who may have had at some previous date an attack of angina, is seized with a severe paroxysm. This passes away, but there is shortly a recurrence, and for several days in rapid succession there are subintrant attacks, with increasing weakness of the heart. Huchard describes the condition as *l'état de mal angineux.* In a way, it is a counterpart of the status epilepticus. The condition is one of terrible distress. I have seen but two cases, and as this feature of the disease has not been specially dwelt upon by writers, except Huchard, I will read you an account of them both.

Case XXXII.—On January 3, 1894, I saw with Dr. Pole, Mr. L., aged fifty-five years, merchant, who for a week had had attacks of severe pain in the region of the heart.

The patient was a stout, large-framed man, who had lived for many years a life of great activity. He had always enjoyed very excellent health; never had had rheumatism. He has seven healthy children. He had been a moderate smoker and moderate drinker, chiefly of beer. He had not had syphilis. Seven years ago, after a slight exertion, he had a very severe attack of pain about the heart, which lasted, however, only a day and then passed off. He had no recurrence and had been very well, though, occasionally, he has been a little short of

breath on walking rapidly. A week ago, December 27th, a fire occurred in his place of business, and he was naturally very much excited, and helped to save the papers and books. That night he had a severe attack of angina pectoris, accompanied with vomiting and sweating. He was better the next day and able to go out. Since then he has had three attacks, all of them of a good deal of severity. He feels very weak and feeble and the pains are severe enough to require morphine. Last night they were very much worse.

He was a well-nourished, healthy-looking man. The pulse was about 90, and there was no increase in tension; the radials were not sclerotic, and though the temporals stood out prominently, they were not firm. During the examination, the patient had an attack of very severe pain, and clasping his hands over the heart rolled about upon the bed. He was flushed in the face, and then broke out into a profuse perspiration. During the attack the pulse did not change materially in character, but remained regular. The pain was described as very intense, a feeling as if the heart was grasped in something. It extended also down the left arm and in very severe paroxysms down the right arm. The apex-beat was difficult to feel on account of the fat mamma. The cardiac dullness was not increased. The sounds were clear at apex and base; the aortic second was not accentuated. The lungs were clear on percussion and the breath sounds were normal.

The abdomen was distended and the stomach tympany was high. As nitroglycerin and nitrite of amyl had no influence whatever on his attacks, morphine was used.

On the 4th he was better. On the 5th and 6th he had very severe attacks, requiring much morphine. On the 7th and 8th he was still worse, and displayed a remarkable resistance to the morphine. Thus, in the hours between ten o'clock Saturday night and 1 P. M. on Sunday, he had received by mouth and by hypodermic injection five grains of morphine, in spite of which he scarcely slept at all, and at the time of the visit, the pupils, though small, were not extremely contracted. So resistant had he appeared to be to the morphine that we discarded the tablets which had been employed and obtained a fresh solution.

The attacks of pain were of great intensity and recurred frequently. They were of the sharp, agonizing form, and in the intervals there was a dull, heavy weight. Only the fullest doses of morphine on Sunday and Monday kept him free from pain. On Tuesday he was somewhat better, and on Wednesday he was almost free.

During these protracted attacks he was frequently almost beside himself with the pain, and sweated very profusely, and on Sunday and Monday and Tuesday he had severe attacks of vomiting. There was no fever. On Wednesday, the 10th, he was better. I saw him early on the morning of the 11th. He had had a bad night with the shortness of breath. I found him with a pulse of 115, small in volume; the heart sounds feeble and distant. The change, so far as his heart was concerned, was very striking, as the heart sounds had previously been quite clear. To-day they were extremely feeble and the action somewhat irregular. Over the left lung there were numerous bronchial râles, particularly in the axillary region. In the evening his condition seemed really critical. The respirations were 40, labored; expiration prolonged, and there were medium-sized râles heard over the whole chest. He was given whisky freely, Hoffman's anodyne, and ammonia, and in spite of the threatening condition in his lungs he was given during the night two or three hypodermic injections of morphine.

On the 12th and 13th the cardiac condition was better. He had had no attacks of pain since Wednesday. The bronchial symptoms and cough continued.

On the 14th he was not nearly so well. The respirations were hurried, the cough troublesome, and over the whole chest piping rhonchi were heard. The pulse was at about 120 and feeble. He took his nourishment better, and the feeling of weight about the heart had gradually diminished. All along, the color of his face had kept pretty good, though that of the finger tips was sometimes a little cyanotic.

On the 15th and 16th he was decidedly better, though the wheezing rhonchi were still present everywhere. His expectoration throughout these attacks had been muco-purulent, and

then purulent, but the cough was never paroxysmal. On several occasions the urine presented slight traces of albumin.

January 21st. For the past few days the condition had been better, little or no pain, less wheezing, and he has been sleeping better and taking more food. Last evening, however, he had hallucinations, and did not know where he was, thinking he was in some hospital, and that his wife was his mother-in-law. He seemed, however, so well that they thought partly that he was joking. His wife stated, too, that on several occasions during his illness he had made odd remarks, as if he did not realize fully his surroundings. He spoke of it himself this morning and joked about it, seeming quite clear and bright mentally. The pulse was soft, 90, regular, and without increase in tension; the heart sounds were a little feeble, but clear. The bronchial râles were still to be heard everywhere over the chest. I left him, saying that as he was so much better I probably would not see him again.

22d. Dr. Pole sent word that the patient died suddenly at 2.45 this afternoon. He wrote: "I saw him about one o'clock, after he had had a severe heart pang, which he described as of a very sharp, cutting character, and he felt as though his heart had stopped. The color changed as usual. He had been cold all day at the extremities, though not more so than he had frequently been before. He rested fairly well last night and took no morphine, but throughout the day he has had cutting pains in his left hypochondriac region."

Case XXXVIII.—On the 24th of February, 1896, I saw, at 10.30 A. M., with Dr. Mary Sherwood, Mr. L., aged fifty-nine years, who had been attacked at seven o'clock in the morning with agonizing substernal pain.

He was a healthy man of good stock; his mother, still living, was aged nearly ninety; his father died about the age of sixty, of, so it is said, fatty heart. The patient had been an abstemious man, of good habits, not a heavy smoker. During the past thirty years he had scarcely had a day's illness. For a year or more he had been using the bicycle, and had noticed that he was a good deal distressed and short of breath on going up hill. For several weeks he has had occasional attacks of pain of a

singular character about the wrists, chiefly the left, which, he said, felt as if encircled by a band. He has occasionally felt pain about the elbow and the left shoulder. They did not seem to be rheumatic. Yesterday he had a very comfortable day, took a light evening meal, and went to bed feeling in his usual health. He was aroused this morning at seven o'clock with a very severe pain beneath the breast-bone. It extended to the region of the apex, and was felt very severely down the left arm and about both wrists. He became pale, but Dr. Sherwood, who saw him about half-past seven, said that the pulse was not much affected. He obtained temporary relief by inhalations of the nitrite of amyl, but between eight and nine it became so severe that he had to be given whiffs of chloroform.

I saw him at 10.30. He was a healthy-looking man, with grayish hair and mustaches; there was no arcus senilis. He was not sweating, and he did not look very greatly distressed. The pulse was 90, of fair volume, without increase of tension, and the coats of the vessel were not specially thickened. The apex-beat was not easily to be felt. The heart sounds were dull and muffled at apex; there was no murmur at the base. The aortic second sound was not accentuated. There was no dullness over the manubrium. He had no respiratory distress, and there were no piping râles. The abdomen was not distended.

The intensity of the pain had passed, but he was still suffering a great deal from a very severe constant pain beneath the breast-bone. He had not had any sweating or special coldness of the hands or feet. He was ordered a quarter of a grain of morphine, and to have it repeated at intervals if necessary. He improved somewhat through the day, though the pain did not entirely disappear. He had a pretty comfortable night.

On Tuesday, the 25th, he seemed better. He had five or six free movements from the bowels, and, as he insisted upon walking to the water-closet, they exhausted him a good deal.

On Wednesday, the 26th, without any active paroxysm, he had a great deal of substernal pain, and his pulse became feebler. He dreaded very much a return of the severe pain, and had small doses of morphine at intervals. I did not see him again until Thursday at 2 P. M. He had not had a good night, and

had become much worse through the morning, signs of great cardiac weakness having appeared. He had had no sweating. When I saw him he was greatly changed. The pallor was marked, and the general depression extreme. There was no sweating; the face was pale, rather than ashy gray. The tongue was thickly furred. His mind was quite clear, and he complained only of feelings of great exhaustion and an uneasy pain beneath the sternum. The head was low; the respirations were not hurried. The pulse was scarcely to be counted, only a few feeble beats reaching the wrist. There was no heaving over the præcordia; the sounds at the apex were only just audible in gallop rhythm. At the base the gallop rhythm could just be perceived. There seemed to be a slight increase in the area of cardiac dullness. He had been having hypodermics of strychnine one sixtieth, but one thirtieth was ordered every two hours, and a hundredth of a grain of digitalin. He had passed very small quantities of urine. At ten o'clock that evening he was decidedly better; the pulse was stronger and the beats were regular. There was still slight gallop rhythm at the apex. The sounds were very much more distinct. He complained a good deal of an unpleasant gasping in his breathing at intervals, which distressed him very much.

February 28th. Patient had had a rather restless night, sleeping only at intervals, and being much distressed by gaspings for breath. He had taken small quantities of nourishment, and had had no vomiting. The pulse was regular, small, and about the same as last evening. He had had digitalin and strychnine regularly through the night. He had a very comfortable day, and seemed altogether better, though he had had some slight delirium and wandering, particularly after waking. He had slept with his head high, and had not been quite so much troubled with the cardiac asthma. He had had no attacks of pain.

29th. This morning he was not so well. He had had a quarter of a grain of morphine at ten last night, which quieted him, but he was aroused at intervals with a distressing sense of the need of air. The delirium was marked and he looked distressed; there was no coldness of the hands and feet, and

no sweating. The pulse was feeble, irregular, and intermittent; sometimes three and four beats were dropped in succession. The apex-beat was not palpable. The heart sounds were only just audible at the apex. There was a gallop rhythm. At the base the second sound could only just be heard. There was no murmur. Throughout the day he was quiet, except for attacks of gasping for breath, which were very distressing. At 5.30 the pulse could not be felt at the wrist. He was conscious; the respirations were not hurried, though every five or ten minutes he would become a little restless and gasp. The heart sounds could be heard both at apex and base; a very distinct embryo cardia, but no murmur. The feet and hands were cold, but he had had no sweating. It was rather remarkable to see a man in such a desperate condition entirely conscious and perfectly alive to his surroundings. He was at times very nervous and restless. Throughout the evening he grew worse; the heart sounds became feebler, and after a period of terrible distress for an hour or more, death occurred, about six days after the onset of the first paroxysm.

LECTURE III.

ANGINA PECTORIS VERA.

PHENOMENA OF THE ATTACK.

Exciting causes.—Symptoms.—State of heart and pulse.—Pericarditis.—Respiratory features.—Gastro-intestinal symptoms.—Nervous and psychical symptoms.

Exciting Causes.—There are three important elements—muscular exertion, mental emotion, and digestive disturbances. Any muscular effort which calls for increased action of the heart is liable to bring on a paroxysm. Heberden refers particularly to this: "They who are afflicted with it are seized while they are walking, more especially if it be up hill." Some patients who can not walk except on the level without bringing on a paroxysm can, however, take active horseback exercise. In extreme cases even an attempt to move in bed or assuming the sitting posture will cause an attack, or such slight exertion as stooping to lace the shoes. Hurrying to catch a train has been often the exciting cause of a fatal attack in the subjects of angina. The muscular and mental excitement of coitus is particularly dangerous, and has in many instances caused death. Two of my patients laid great stress on the terrible character of the attacks which had followed the act.

The well-known effect of mental emotion has never been better expressed than by John Hunter, who used to say that "his life was in the hands of any rascal who chose to annoy

and tease him." And yet some of the victims of angina have not found mental excitement to be the most serious exciting cause. Thus, in Mr. Sumner's case, "a sudden turn in his easy-chair, while quietly reading at night, would start up the most tearing agony, while at other times an exciting speech in the Senate, accompanied with the most forcible and muscular gesticulations, would not create even the suggestion of a pain."—(Taber Johnson.)

For some of the worst attacks, however, neither muscular action nor mental emotion is responsible, since they come on when the patient is quiet and at rest, or may wake him from sleep. Cold is another exciting cause, particularly in the vaso-motor form, but in the organic variety a cold wind, even the opening of a window in winter, or the cold sheets at night have been known to bring on an attack.

In almost every case in which the paroxysms recur with frequency the patient lays stress upon the condition of the stomach. Exertion immediately after a full meal, the eating of certain articles of food, and especially of late suppers, are very apt to cause attacks; and, as I will mention later, there are instances in which the dyspepsia is so marked a feature that the character of the disease is entirely overlooked. In some patients flatulency is one of the most common exciting causes.

Symptoms.—In the report of the two cases which I read to you at the end of the last lecture I described the phenomena associated with severe attacks. The physician has not often an opportunity of watching the onset and course of a paroxysm. Only once that I remember did a patient have an attack in my consulting room, Mr. S., to whose case I have already referred (XXVI). As he sat quietly in the chair, just after the completion of my examination, his eyes became fixed and he suddenly grasped both hands over the heart. For a moment the face did not change; then it flushed, and the neck became

swollen, and the cervical veins full. The face became very much congested, and tears filled the eyes. The respirations, which had been 18, increased to 30 in the minute. The pulse, which had been 80, increased to 90, and became smaller and harder. Considering the increase in the respirations, and the congested state of the face and neck, I was surprised that the pulse changed so little. He remained immobile during the entire attack, which lasted just a minute and a half, passing off abruptly, and he at once began to put on his clothes.

There are two chief elements in the paroxysm: first, the pain—*dolor pectoris;* and second, the indescribable feeling of anguish and sense of imminent dissolution—*angor animi.*

The resources of the language have been taxed to describe the pain of angina pectoris. Patients speak of a hand of iron grasping the heart, or a band of metal encircling it and being gradually tightened; or as though an enormous weight was compressing the breastbone against the spine, or as though the whole chest were compressed in an iron case. In other instances the pain is associated less with pressure than with the sensation of stabbing, as though a dagger had transfixed the heart. While the maximum intensity of the pain is substernal (whence the name of *sternalgia* is derived), it may be in the upper or lower part of the breastbone, or over the body and apex of the heart. There are cases in which the chief agony is opposite the point of the xiphoid cartilage in the scrobiculus cordis. During an attack there may be marked tenderness over the region of the heart, or the left breast or the nipple may be tender to the touch. The pain may cease as abruptly as it began. One of Parry's patients said the transitions from acute pain to a state of ease were so sudden that at times he felt both extremes at the same moment.

A feature noted by Heberden and all the early writers was the radiation of the pain to other parts. Heberden says:

"It likewise very frequently extends from the breast to the middle of the left arm. . . . The pain sometimes reaches to the right arm as well as to the left, and even down to the hands, but this is uncommon. In a very few instances the arm has at the same time been numbed and swelled." In an instance reported by Heberden the patient had attacks of pain in the left arm without any affection of the chest for fifteen years prior to his sudden death. The pain most commonly extends to the shoulder, to the left upper arm, and to the neck of the same side. When it extends to the arm and hand it is along the inner surface of the upper arm, and in the lower arm on the ulnar side in the distribution of the ulnar nerve. The feeling is one of numbness and tingling, or of pins and needles. There may be hyperæsthesia of the skin. Very often the chief pain is in the region of the elbow, or there may be, as in a case I have already narrated to you, a band-like sensation around the wrist. Sometimes the radiation of the pain is more marked in the right arm and in the right side of the chest. Quain states that Dr. Morison has reported a case in which disease of the right side of the heart was accompanied by symptoms of angina affecting the corresponding side of the chest and arm. Curiously enough, as noted by Heberden, the pain in the arm may precede the angina attacks for years. Blackall, in the interesting appendix upon Angina to his work on *Dropsies*, refers to the account which Lord Clarendon gives of his father's sudden death, evidently from angina, "without one minute's warninge or feare," though the pain is said to have been only in the arm. As this case is often referred to, I will give you the extract from the *Life*. Mr. Hyde was in church, and "found himself a little pressed as he used to be." Going to his home, "the pain in the arm seizing upon him, he fell down dead, without the least motion of any limb." In some cases there is sen-

sory disturbance throughout the entire left side, a feeling of numbness or tingling in the neck, arm, and leg. There are instances on record of extension of the pain to the left testis, with swelling; or the attack may begin with furious pain in this organ.

There are very interesting areas of cutaneous hyperæsthesia in the attacks, chiefly in the præcordia, about the pectoral fold, and sometimes along the side of the neck. They have been studied particularly by Mackenzie, and are rarely absent.

I do not know of any clearer view in explanation of the radiation of the pain in angina than that which was afforded by the late Dr. James Ross, of Manchester. I will quote a brief summary. I do not know whether it was ever elaborated. "When a viscus was diseased there was local pain which might be regarded as of splanchnic origin (præcordial pain in the case of the heart). In addition, the irritation was conducted to the portion of the spinal cord from which the viscus derived its splanchnic nerve, and thence spread in the gray matter of the posterior horns, whence by the law of eccentric projection it was referred to the termination of the somatic nerves derived from the segment of the cord—the second and first dorsal in the case of the heart. This explained the pain shooting between the shoulders and down the inner side of the arm (second dorsal) to the elbow and the ulnar border of the forearm and hand and ulnar fingers (first dorsal)." *

The subsequent studies of Mackenzie and of Head have fully corroborated this view. Head † concludes that:

"1. In diseases of the heart, and more especially in aortic disease, the pain is referred along the first, second, third, and fourth dorsal areas.

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* *Lancet*, 1891, i. † *Brain*, xvi.

" 2. In angina pectoris the pain may be referred in addition along the fifth, sixth, seventh, and even the eighth and ninth dorsal areas, and is always accompanied by pain in certain cervical areas."

A very remarkable feature is the motor disability which may follow a severe attack. The left arm may not only be numb, but for a time almost powerless. Blackall says that he has seen instances in which the muscles of the arm and chest were not only painful, but were affected with a twitching noticeable by the patient, and visible to the observer. B. W. Richardson * says " the voluntary muscles seem to be affected and rigid." Still more extraordinary is the fact, noted by Eichhorst, † of atrophy of the muscles of the hand supplied by the ulnar nerve.

Von Dusch, in his admirable *Lehrbuch der Herzkrankheiten* (which remains one of the best works of its kind in the literature), refers the hiccough, the occasional difficulty in swallowing, the globus and uneasy feelings in the throat, and the gastric symptoms to sympathetic involvement of the phrenic and vagus nerves.

Vaso-motor disturbances are almost constant in the attack. A sudden pallor of the face may be the first indication, and, as a rule, vaso-constrictor influences prevail in the severe paroxysms. A cold sweat breaks out upon the forehead and upon the arms and legs. In recurring attacks I have seen the skin of the hands like that of a washerwoman from constant soaking in perspiration. As in Case XXXV, there may be great pallor and coldness without sweating. Though rarely absent in the organic form of the disease, these vaso-constrictor disturbances are often more pronounced in the hysterical angina. The countenance is expressive of the deepest anguish,

* *Asclepiad*, vol. xi.

† *Handbuch der speciellen Pathologie*, 5te Auflage.

and may assume a deathlike, ashen hue. In other instances, as in Case XXVI, the face is suffused, or even deeply congested at the outset, and the veins of the neck may stand out prominently. More commonly in a fatal paroxysm there is pallor at first, which is followed by great lividity, as noted by Powell * in a man who died in his consulting room.

Complaints of coldness and of swelling of the extremities are more frequent in the hysterical form.

In many cases of true angina the pain alone is experienced, but in severe paroxysms the other factor—the mental element, the *angor animi*—is also present. Latham was the first to distinguish clearly these two features of the attack: "The subjects of angina pectoris report that it is a suffering as sharp as any that can be conceived in the nature of pain, and that it includes, moreover, something which is beyond the nature of pain—a sense of dying." And he adds, "the dying sensation I have more frequently found to surpass the pain than the pain the dying sensation." The one is in reality a physical, the other a mental phenomenon, and was described by Heberden's unknown correspondent as the sensation of a universal pause in the operations of Nature, or a sense of imminent and immediate dissolution. This feature of the attack was certainly referred to by Seneca (quoted by Gairdner) when he says, "As compared with any other disease, it is like the difference between being sick merely and giving up the ghost." Associated with this sensation there may be a feeling of air-hunger, or, as one patient expressed it to me, the same sensation that one has after holding the breath for as long as possible; yet the attack is not necessarily associated with any special respiratory disturbance.

The *attitude* during an attack is best described by the

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* *Practitioner*, vol. xlvi, p. 254.

word immobile. If seized on the street, the patient grasps a lamp-post or leans against a wall, unable to stir until the agony has passed off. The attack usually comes on during some slight exertion, while the patient is in an erect posture. He may be quite unable to sit down. In other cases, when the attack comes on at night, the patient usually assumes the sitting posture, or he finds slight relief by pressing a firm pillow to the chest, or by pressing firmly against the back of a chair. Immobility, however, is not a constant feature of a paroxysm of true angina. In Charles Sumner's case, Dr. Taber Johnson notes that he would at times get ease by walking the floor, quite unconscious of any increase in the agony by the exertion. In others the erect posture is assumed with the head and shoulders thrown back. One patient assured me that in moderate attacks on the street, by a strong effort of the will, he could continue to walk and the pain gradually subsided. This is like the gigantic farmer, of whom Forbes tells, who thought he could rule the disease as he did his horses.

State of the Heart and Pulse.—Heberden states that "the pulse is at least sometimes not disturbed by this pain, consequently the heart is not affected by it." Parry is more positive as to the occurrence of change, holding that "whatever may be the state of the pulse as to regularity, I believe we shall always find it become more or less feeble according to the violence of the paroxysm." The question is one about which very diverse opinions are held, and you will find in vol. i of the *Lancet*, 1891, several interesting letters which passed between Professor Gairdner and Dr. Harrington Sainsbury. It is quite evident that there are good authorities who accept the statement that in some cases at least the paroxysm is not associated with special change in the pulse, and consequently not in the action of the heart.

The opportunities for observing the paroxysm do not come very often, and when they do the condition of the patient is such that our efforts are directed rather toward his relief than to the study of special points in the case. In an attack of moderate severity, such as Mr. S. (Case XXVI) had in my consulting room, the pulse, which had been 80, increased to 90 in the minute, and became smaller and harder. The tension certainly became increased, but I had not time to do more than count the radial beats for half a minute and to listen hurriedly to the heart sounds before the attack was over. In Case XXXII, in the first paroxysm in which I saw him, January 3d, the state of the pulse threw me a little off my guard; it was full and regular, and did not change much, if at all. I am not certain that it was an intense attack, as he threw himself about on the bed, the face was flushed, and there was a good deal of commotion. Subsequently the pulse became feeble and irregular, 115 a minute. Then, on the day before his sudden death, the pulse was soft, regular, without special tension, and 90 a minute. In Case XXIII the pulse fell in the paroxysm to 42 in the minute and became small and soft. For days the range had been about 96. For several hours after the paroxysm the beats at wrist and at heart ranged from 40 to 50 a minute. Subsequently the heart beats became more numerous than the pulsations at the wrist, ranging from 60 to 70 a minute. In Case XXXVI I did not see the patient in his first paroxysm, but three hours later the pulse was 90, of fair volume, regular, and without increase of tension. On succeeding days, as the attacks increased in frequency, the pulse became small, feeble, and at times could not be felt. Following a series of severe attacks, the pulse may be persistently small and irregular, as in Case XXXV. In Case IV, that of a man, aged forty-five, admitted to the University Hospital, Philadelphia, February 24, 1887, I had

several opportunities of feeling the pulse during the paroxysm. On the 25th the pulse was 80, regular, and small, and the respirations 34. During an intense paroxysm the pulse became more and more feeble and at last *could not be counted.* This sentence I find underlined in my notes.

Osgood has called attention * to a remarkable difference in the radial pulse of the two sides. The case was one of hysterical angina in a young girl. Huchard (p. 524) refers to its occurrence in true angina, both in the attacks and in the intervals. The heart's action in severe spells is probably always disturbed, the force of the impulse weakened, and the rhythm altered. There are two changes which have been most common in my experience—namely, the shortening of the long pause and the occurrence of gallop rhythm. Whatever may be the mechanism of the production of these changes, they both, I think, mean the same thing, weakening of the ventricular systole from dilatation, and debility of the muscular wall. The case which called my attention to the fœtal heart rhythm following angina I saw with Dr. Underwood, at Pittston, Pa., in February, 1889. The patient, Case VI, aged sixty, had well-marked signs of myocarditis, with cardiac asthma and severe pains about the heart and down the arm, so that he had to take morphine freely. I saw him shortly after an attack; the pulse was 104, weak, and irregular. At apex and base the sounds were clear, rather ringing in quality, and all distinction between the two seemed lost. "There was a shortening of the pause between the second and the first sounds, so that they followed each other in a uniform series, as in the fœtal heart beat." This, so-called, embryocardia was a most persistent feature in Case V, and was present also in Cases XIX, XXIX, XXXV, and XXXVI. The gallop

* *American Journal of the Medical Sciences*, October, 1875.

rhythm is, I think, met with quite as often, and was present after attacks in Cases XI, XIII, XIX, XXXII.

It does not fall to the lot of many physicians to witness a sudden death in angina, but there are observations to show that the pulse beats (and the heart) stop abruptly. Potain mentioned a case to Huchard (p. 525), and in the case of our good friend, Mr. E., Case XXXV, Dr. Thayer, who was present, tells me that the death seemed instantaneous—the pulse ceased *at once*, and there were no further heart beats. (Note C.) As I before remarked, the mode of death resembles that produced by Kronecker's heart puncture.

As the subjects of angina pectoris present very frequently the signs of arterio-sclerosis and increased tension, you will often find a ringing, accentuated, aortic second sound. An aortic diastolic murmur is much more common than my figures would indicate. As I have already mentioned, mitral-valve disease is rarely present. There is a very interesting feature in certain cases of angina with recurring attacks—viz., that with the development of a mitral systolic murmur the attacks have ceased as though a relief of the intraventricular pressure had been effected by the establishment of a relative mitral insufficiency. My attention was called to this point by Musser,* who has had several illustrative cases, and Broadbent has dwelt particularly upon this point.†

Pericarditis.—During a severe attack pericarditis may develop from the involvement of the epicardium in a softening infarct (Kernig). ‡ Dock # has described the onset of pericarditis in a case of thrombosis of the coronary artery, due to the same cause. Hood || records a case in which the fric-

* *Transactions of the Association of American Physicians*, x, p. 85.
† *British Medical Journal*, 1891, i, p. 747.
‡ Quoted in *Lancet*, August 20, 1892.
# *Medical and Surgical Reporter*, 1896.
‖ *Lancet*, 1884, i, p. 205.

tion developed twenty-four hours after the attack, and subsequently there were signs of effusion. In the discussion which followed, De H. Hall mentioned a similar case.

Respiratory Features.—We have here to consider several important points—the symptoms in the attack, the relation of cardiac asthma to angina, and the interesting group of cases of chronic pleuro-pulmonic affections in which angina-like attacks of great intensity occur.

(*a*) In the attack, except slight acceleration in the movements, there may be no special changes. You will remember, in reading John Hunter's case, that, as he expressed it, he felt as though he had forgotten to breathe; and a patient may feel some sort of relief from the pain by voluntarily fixing the chest at the full inspiration, or by making a very forced expiration and holding the breath. In a lethal attack the respiration may become slowed and sighing, and a few gasps follow the abrupt cessation of the heart's action. One of the most remarkable features of the attack to which the attention was early called is the development of a bronchial asthma. Erasmus Darwin * called the disease painful asthma—*asthma dolorificum*—without, so far as I can see from his account, any justification. On auscultation one hears over the chest numerous sibilant râles, and the breathing may become labored and expiration much prolonged. Huchard likens it to a condition of acute emphysema. In Case XXXII, which I gave you in full at the last lecture, the attacks of shortness of breath with piping râles formed a very distressing feature in the case. The expectoration was muco-purulent; Curschmann's spirals were never found. Throughout the illness, which persisted for several weeks, this condition continued, and was the cause of much annoyance. Though Heberden

* *Zoonomia*, third ed., 1801, p. 41.

does not refer specially to the asthma, he speaks of two patients who had spat up blood and matter. Many patients have referred particularly to the "wheezing" which has accompanied the attacks. Goodhart,* who describes the condition as an acute bronchitis, thinks it of very grave prognosis. The same bronchial wheezing is present in some cases of cardiac asthma and doubtless gave the name to this symptom.

(*b*) Cardiac asthma may develop during an attack or alternate with the paroxysms of pain. In another lecture I shall speak more at length on the relations of this feature to angina, particularly to the angina pectoris *sine dolore*. Here I wish only to call your attention to the distressing spells of dyspnœa, chiefly nocturnal, which may come on in the subject of angina, either independently of or following an attack. In the cases with advanced arterio-sclerosis the cardiac asthma may be the most pronounced and distressing feature, disturbing the patient at night, making him dread to fall asleep, owing to the horrible sensations which accompany the awakening in a paroxysm of dyspnœa. The subject may die in an attack of angina after a long series of asthmatic seizures. Case XXIII, Dr. ——, aged forty-seven years, from Santa Fé, N. M., had advanced arterio-sclerosis. Fifteen months before his death he had an attack of angina; then for a year he had many attacks of cardiac dyspnœa, chiefly nocturnal, and once had transient hemiplegia with aphasia. He died after several paroxysms of terrible angina, recurring in the course of twelve hours.

Cardiac asthma is an everyday symptom in the course of chronic valve disease and cardio-sclerosis. In hospital practice it is as common as angina pectoris is rare. It may recur in paroxysms very like angina pectoris, in one of which the

* *Guy's Hospital Reports*, vol. xliv.

patient may die. Dreschfeld reports * the case of a woman, aged forty-nine years, who, when younger, had been hysterical, and later very neurasthenic. Suddenly, one night she was seized with severe dyspnœa, without any cardiac pain. A week later she had a second attack, again without pain, and in a third attack, the following night, she died. There was a fibrous myocarditis at apex of left ventricle, and the left coronary artery was greatly narrowed by an atheromatous plate.

Cheyne-Stokes breathing is met with in the intervals between very severe attacks, as in Cases XXIII and XXXII, or is one of the manifestations of an advanced arterio-sclerosis, as in Cases V, XI, and XIII.

(*c*) The term *respiratory form* of angina pectoris has been applied to cases of cardiac asthma, such as the one reported by Dreschfeld. I think the term more appropriate to that interesting group of cases in which the subjects of chronic pulmonary or pleural disease have agonizing paroxysms of pain about the heart, evidently of the nature of angina, and which may prove fatal.

Let me mention several illustrative cases:

Case IX.—On February 12, 1891, I was consulted by a healthy, vigorous-looking man, aged thirty-three years, who complained of shortness of breath and attacks of agonizing pain in the chest.

In 1876 he had pleurisy on the right side, for which he was tapped repeatedly. The effusion becoming purulent, opened spontaneously, and the fistula took a long time to heal. He gradually got strong and well, and remained so for nearly ten years. In 1887 he began to have attacks of shortness of breath at night, with pain in the chest. At first there was no shortness of breath during the day except on active exertion. In

* *Practitioner*, vol. xliv.

the year 1888 the attacks recurred at intervals. In 1889 and 1890 he was very much incapacitated by them, as there was great pain and shortness of breath on attempting any extra exertion. The attacks came on with a feeling of great oppression in the chest and a sense of overpowering constriction and uneasiness in the region of the heart. The pains never extended down the arms, but they passed up the neck to the head. Of late they have recurred at night with great regularity, so that he dreads to go to bed. He goes to sleep quietly, dreams a good deal, but always, prior to waking in pain, there is great excitement in the dreams, and he feels pressure on the eyeballs and forehead, which gradually increases until it awakes him. Then he arouses in terrible agony in both the chest and head, and the sweat pours from him. The paroxysm lasts from five to ten minutes, and he has often had to take chloroform. During these nocturnal attacks there is no shortness of breath, only the agonizing pain in the region of the heart and passing up the neck to the head.

On examination the patient showed an extreme grade of contraction of the right side, with lateral curvature of the spine, flattening in the mammary and axillary regions, with scarring in the seventh, eighth, and ninth spaces, where the empyema had perforated. On percussion there was flatness everywhere over this side; the left side was hyperresonant. The apex-beat was not visible; the heart impulse could be felt with moderate force at the lower sternum. The heart sounds were perfectly clear and quite natural. The pulse was regular; the vessels were not sclerosed. There was no tracheal tugging, and the manubrium was clear on percussion. On rapid exertion the face became a little flushed, but no murmur developed over the heart. The urine was clear. The abdomen was distended, the right costal border was curiously everted from contraction of the chest, and the liver was drawn up very far. A short time after his visit to me the patient died suddenly in a paroxysm.

The following case illustrates a much less severe form:

A clergyman, aged forty-four years, came under my care April 20, 1892, with signs of local disease at the apex of the

right lung. He was a vigorous, wiry looking man, who had had tuberculosis for several years; but the feature which incapacitated him for work was the occurrence during excitement, and especially when preaching, of attacks of indescribable distress about the heart, which on several occasions almost caused him to faint. It was not a sharp pain, and there was no radiation, but he described it as a feeling as though the heart would burst or break, and an entire impossibility to proceed with his sermon, or with his address. It was not accompanied with any shortness of breath, and though the signs of tuberculosis and of some compensatory emphysema were quite marked, yet it was this special symptom for which he sought relief, as by it he was incapacitated. The apex-beat was not visible. The heart sounds were clear; there was no sign of hypertrophy, and the aortic second sound was not accentuated. The arteries were sclerotic, and the pulse tension was considerably increased.

The condition which this patient described, though probably not true angina, is of interest in connection with this subject. Public speakers and others who have to address audiences not infrequently complain of a peculiar sensation in the region of the heart, sometimes only an exaggeration of the ordinary embarrassment which so many of us feel, but in other instances there may be, with an increased peripheral vaso-motor contraction, quite evident in the pallor of the face, a summation of cardiac distress which becomes almost unbearable. I know of one professional friend who rarely can get up to speak in a meeting without considerable cardiac pain.

Cases of chronic pleurisy, tuberculous or otherwise, are very apt to have severe angina-like attacks. I have called your attention in the wards to Mary C., aged twenty-four years, with chronic bilateral pleurisy, who came under our care first in December, 1890, with an effusion on the right side. She has had lately severe attacks of pain in and about

the heart which have come paroxysmally, but have never had the intensity of true angina. I saw this winter a woman, aged thirty-six years, who had had a pleurisy on the right side of thirteen years' duration, with chronic disease of both apices, and considerable enlargement of the heart. She had had shortness of breath, and occasional pain about the heart on exertion. She died in an attack of acute dilatation of the heart, associated with a great deal of substernal pain, much pallor, and sweating.

Gastro-intestinal Symptoms.—Nausea not infrequently accompanies the attack, and the patient may vomit. Heberden notes that "persons who have persevered in walking till the pain has returned four or five times have then sometimes vomited." As an attack ends the patient may belch quantities of gas, or pass flatus from the bowel, both with apparently great relief.

Flatulency was regarded by Butter as "the most obvious and the most regular exciting cause." Parry, too, laid great stress on the influence of eructations in mitigating the pains produced by "mal-organization of the large vessels," and quotes Morgagni to the effect that the vulgar, and even the physicians, thought the disease originated in the flatulency. There is another important relation of the gastro-intestinal features of angina pectoris. When the pain is situated in the scrobiculus cordis, and associated with eructations and dyspepsia, the diagnosis of gastralgia may be made. There are several very interesting papers on this question in the literature. Leared * described a series of cases of "disguised disease of the heart" in which the "heart affection was so strangely masked by that of the stomach that nothing in the statements of the patients had any bearing on the primary

* *Medical Times and Gazette*, 1867, i.

disease." In several of his cases sudden death followed. Barié * has written an elaborate paper on the cardio-pulmonary features of gastro-hepatic disorders. The attacks of pain in some of his cases simulated closely angina. Huchard has a special section on what he called the *pseudo-gastralgic* form of angina.

In only two cases in my series were the gastric symptoms of such intensity that the affection was at first thought to be in the stomach.

In Case XIX the pains were at first altogether in the upper part of the abdomen, and as they were of sufficient intensity to cause vomiting she was thought to have gastralgia. It was not until dyspnœa came on, and the pains became centred about the heart and extended to the neck and arm, that the diagnosis of angina was made.

In the following case the patient, a most intelligent man, insisted that the entire trouble was in his stomach.

Case XXVII.—Mr. W., a merchant from North Carolina, was referred to me by Dr. Whitehead, October 26, 1893, complaining of severe attacks of pain in the abdomen and lower part of the chest. He was sixty-seven years of age, and of excellent family history. All his life he had had occasional attacks of indigestion.

On the 17th of June, 1893, after helping a servant to carry a heavy trunk upstairs, he felt a sensation at the pit of the stomach as if he had wrenched himself badly. A few days later, while walking up a hill, the sensation of pain in the stomach returned, and a week later, while walking fast to catch a train, he had very severe pain and shortness of breath. The taking of food apparently made no difference to the pain, but he had a good deal of belching of wind, and he insisted upon regarding the condition as altogether due to his stomach. Dr. Whitehead, in describing the case, stated that the pain began in the

* *Revue de médecine*, 1883.

epigastrium and passed directly to the backbone; if very severe, it spread over the thorax; "asthma comes on; there is tingling sensation in the left hand, and violent pains are felt in the arms. The agony is simply terrific." He never had any nausea or vomiting in the attacks. The patient was a very well preserved man; the radials were firm, tension was increased, and the radial pulse was anastomotic. There was no excessive cardiac impulse, the area of dullness was not increased, but the subcutaneous fat was very excessive. With the exception of a soft apex systolic murmur, auscultation gave no indications. The second sound over the aortic region was of medium intensity. The examination of the abdominal organs was negative. There was no dilatation of the stomach and the gastric juice was normal. The note which I made with reference to the nature of the case at the time was as follows: "Though the possibility has been entertained that Mr. W. has gastralgia, due either to ulcer or cancer, it seems to me much more likely that he has angina pectoris." He was ordered iodide of potassium; and throughout the winter of 1893–'94 he did very well, and he could walk a distance of two or three blocks without suffering pain.

On July 2, 1894, after eating a much heartier dinner than usual, he went out to pay a visit, and on leaving the door of the house fell forward on the verandah and died in a few moments.

Another by no means uncommon and often very distressing symptom is persistent hiccough.

A symptom described by some writers has been the constant desire to urinate—urina spastica—and there may be a very large amount of urine passed. Griffiths and Massey * profess to have separated a special leucomaine from the urine passed during the attack.

Nervous and Psychical Symptoms.—The mental anguish has been described. A sense of faintness almost invariably

* *Comptes rendus, Acad. des sciences*, 1895, 1128.

accompanies severe attacks, and actual loss of consciousness, syncope, may follow, upon which feature I have already dwelt. There are interesting psychical manifestations in angina pectoris upon which you will not find much in the literature. They are features of the myocarditis, rather than of the angina, and develop with the progressive weakness of the heart. In Case V the patient had delusions of a most painful nature for nearly six weeks, during which time he had an exceedingly feeble heart, with gallop rhythm. He recovered and lived for three years. In Case VIII the patient thought that he was in a strange house and begged constantly to be taken to his home. You remember that in Case XXXII there were occasional delusions.

Trousseau believed that there was a close relationship between angina pectoris and epilepsy: "In some cases, and perhaps in a pretty good number of instances according to my experience, angina pectoris is an expression of this cruel and fearful complaint, and is a variety of the vertiginous form of the disease—in other words, it is an epileptiform neuralgia. Its invasion is as sudden, its progress as rapid, and its disappearance as sudden, and, as I have already told you, it is not of very uncommon occurrence to find persons who have in former years suffered from angina pectoris become subject afterward to epileptic fits, just as in other instances angina pectoris has been preceded by well-marked epileptiform seizures." * Quite recently Richardson † has urged that angina pectoris is a special disease, of a paroxysmal nature, as distinct as epilepsy and partaking in many ways of its features—a sort of epileptic counterpart in the sympathetic system. The two diseases may co-exist. We have to distinguish between the attacks of nervous palpitation with

* *Clinical Medicine.* New Sydenham Society edition, vol. i, p. 662.
† *Asclepiad*, vol. xi.

cardialgia in epileptics, not infrequent symptoms, and attacks of true angina. The only instance of combination of the two disorders which I have met is the following:

Case III.—An engraver, aged forty-eight years, was admitted under my care to the University Hospital, Philadelphia. He had served in the army during the war of secession, and entered the navy as a marine in 1871. After a blow on the side of the head, in the latter part of 1873, he was insane for several months and required constant watching. He recovered, but has had ever since, at intervals, epileptic attacks, and he has frequently been picked up unconscious on the street. For the past four years he has had also violent pains in the chest with choking sensation, difficulty in swallowing, and shooting pains down the left arm. He does not lose consciousness during these attacks, but they are evidently of terrible severity, and he feels in each one as if he were about to die. He has a well-marked aura preceding the epileptic fit, which starts in the lower part of the chest, but he is not aware of any close association between the epilepsy and the attacks of angina. The patient was in a very bad condition on admission, almost pulseless at the wrist, but after the administration of whisky and digitalis he revived, and in a few days seemed quite himself again. He had hypertrophy of the heart, with aortic insufficiency.

There was in the wards last June (1895) a colored man, aged thirty-four years, who had remarkable attacks of pain about the heart with unconsciousness. He was a healthy-looking fellow; the pulse was not slow, the tension was increased, and the radials felt a little hard. The heart was not enlarged; the aortic second sound was a little accentuated. The urine was normal. He had probably had syphilis. Eight years ago he began to have pains about the heart, and from July to September the attacks were so severe that he was unable to work. In December they returned, and ever since, at intervals, he has been subject to them. Any extra exertion,

such as walking fast up hill, or mental excitement will cause severe pain, exactly under the left nipple, often of great severity. In March of this year, while working in a stable, he felt a sudden agonizing pain in the heart, became giddy, and fell to the floor unconscious. He did not bite his tongue, and, so far as we know, he did not "work" the muscles or foam at the mouth. On June 17th he had a second attack, with very much more pain about the heart, which lasted for five or ten minutes before he became unconscious. On the 24th he was walking on the street, felt a severe pain and great oppression about the heart, and then fell unconscious and was brought to the hospital by the police patrol. The loss of consciousness lasted several hours. He had no attacks while in the ward, and it seemed impossible to determine precisely the nature of the case—whether the so-called cardiac epilepsy, or an anomalous type of angina pectoris. Newton * has reported an interesting case in which very probably both the epilepsy and the angina were associated with syphilis.

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* *Medical Record*, 1893, i.

LECTURE IV.

ALLIED AND ASSOCIATED CONDITIONS.

I. Syncope anginosa.—II. The Adams-Stokes syndrome.—III. Angina sine dolore.—IV. Cardiac asthma.

I WISH to call your attention in this lecture to several interesting conditions closely allied to true angina which may either develop in the course of an attack or which occur spontaneously in the subjects of heart disease or arterio-sclerosis.

I. *Syncope anginosa.*—You remember that Parry called angina *syncope anginosa*, and this feature of *faintness* may detain us for a few moments. The distinguished old Bath physician, from whose monograph I have so often quoted, says: "The angina pectoris is a mere case of syncope or fainting, differing from the common syncope only in being preceded by an unusual degree of pain in the region of the heart." This is too strong a statement, as in a majority of the paroxysms, though the pallor and other vaso-motor phenomena of a *faint* may be present, consciousness, unhappily for the poor victim, is not lost. In looking over the histories of my cases I do not find *fainting*, as we usually understand the term, to have been a common symptom. There is, of course, the syncope of a fatal paroxysm—*S. letalis*, as Quain terms it. During a severe attack the patient may lose consciousness. Mr. S., Case XXVI, was once picked up on the street. In Case XXV, mentioned in connection with angina and epilepsy, we could not determine the nature of the attacks of loss

of consciousness. Another feature of which I have no illustrative example is thus referred to by Broadbent: * A patient who has ceased to suffer with attacks of angina "may have attacks of what he calls faintness, in one of which he ultimately dies. These which have lost the title to the name angina have an equally serious significance." And, lastly, an individual subject all his life to fainting spells may present remarkable attacks of the nature of Gairdner's angina pectoris *sine dolore*, about which I shall speak shortly.

Case XXXIV.—T. J. J., aged sixty-one years, seen with Dr. King, May 11, 1895, complaining of curious attacks which occur on the street while walking.

The patient has been a very vigorous, healthy man, has never had syphilis, and has been abstemious. He has had two attacks of sciatica in the past ten years, the last, a severe one, two years ago; he has had no joint affections. He has had an exceptionally healthy life. From boyhood, however, he has been liable to faint on very trifling provocation, such as a vomiting attack, a slight shock, the sight of blood, or the extraction of a tooth. From any of these causes he would drop instantly in a faint. He has not had a spell of this kind for more than two years. His present attacks date from eighteen months ago. The first one occurred when walking from the Union Station to North Avenue. He had a tingling feeling in the hands, and then a sudden fainting sensation, as though he was going to die. *He had no pain.* The attack passed off in a few moments. He took the street car and then walked to his home, having a second attack on the way. Subsequently he had these attacks at intervals, always when walking on the street.

On November 22, 1894, he had two very severe attacks, and he then consulted Dr. King. In every instance they have come on while he is walking. He does not think that going up hill or walking against the wind makes any difference. He has never had an attack at his place of business or in his home, and he

* *British Medical Journal*, 1891, i, 747.

is able to go up three or four flights of stairs quickly and readily without the slightest embarrassment. They come on with abruptness, begin now every time with a feeling of numbness and tingling in fingers and hands, which sometimes extends up the arms, and which is not more on one side than the other. He has never vomited in an attack; there is no cough, and there is no dyspnœa. He turns of an ashen-gray color, sweats profusely, and feels in each one as though he would sink away and die. It is this sensation of impending dissolution which has alarmed him so much. He has never had the slightest sensation of pain. During an attack he is not immobile, but he has to move slowly. The day before yesterday, for example, he had an attack before he reached his house, and was able to get up the steps into the porch and close the door; but he had then to sit down, and he was found there by his son in a condition of exhaustion and sweating profusely.

He was a healthy-looking man, with iron-gray hair and moustache; no arcus; the pupils were normal. He was not stout, but well nourished. The pulse was 72 and regular, the vessel wall not specially sclerotic, and the pulse could be compressed readily.

There was a slight throbbing in the vessels of the neck. The venules were marked along the course of the diaphragm. On auscultation there was a short, sharp, somewhat rough murmur heard only in the apex region and as far as the mid-axilla. The apex beat was not visible, but was palpable in the normal situation, in the fifth space just below the nipple. The heart impulse was felt also below the ensiform cartilage; there was no thrill. The dullness began on the fourth costal cartilage and did not extend beyond the nipple line. The percussion on the manubrium was clear. The aortic second sound was not accentuated. Both sounds were clear in the vessels of the neck; the second was a little loud at the sternal notch.

The lungs were clear. Posture made no difference in the heart sounds or in the apex murmur.

The liver was not enlarged; spleen not enlarged.

After dressing, and in the erect posture, the pulse was 88 a minute.

June 14, 1895. I heard of this patient to-day. He has had no attacks for a month.

May 29, 1896. The patient was seen to-day. He had a severe attack in April of this year, one of the worst he has ever had. After a hearty dinner he was attacked in the street. There was no shortness of breath, but an "all-gone" feeling, as though he were going to expire, but there was no pain with it; sweat "rolled off" him. He was well that evening. He has had in the year about eight mild attacks. He had an attack yesterday. They occur nearly always after meals.

II.—*The Adams-Stokes Syndrome.*—There is a most interesting group of symptoms associated with myocardial changes, and sometimes with angina, to which Robert Adams, of Dublin, first called attention, and which Stokes subsequently described more fully. Most of the text-books refer to a pseudo-apoplexy in connection with fatty or fibrous myocarditis, a condition in which with a permanently slow pulse the patient has transient vertigo, or falls into a deep coma, with or without convulsive movements. Huchard has given it the name *maladie d'Adams* or *Stokes-Adams.* As it is always pleasant and profitable to have the author's first-hand description of any symptom or disease, I will give you an abstract of the case recorded in the *Dublin Hospital Reports*, vol. iv. Adams, I may remind you, was one of that distinguished band of men, including Cheyne, Colles, R. W. Smith, Graves, Stokes, and Corrigan, who gave such renown to the Dublin school in the first half of this century. He is best known through his superb work on rheumatoid arthritis. Adams's patient was a man, aged sixty-eight years, who had had in seven years not less than twenty apopleptic attacks, each of which was preceded for a few days by hebetude and loss of memory. The pulse was permanently slow, and at the time of the attacks became slower. There was never any

paralysis. Death followed an attack. Post mortem, the heart was found to be excessively fatty. There was no note about the coronary arteries. R. W. Smith * also noted the condition of very slow pulse with fatty heart, and Stokes described it more fully,† and suggested the name false or pseudo-apoplexy. He laid stress on the syncopal character of the attacks, their frequency, the absence of paralysis, and the good effect of a stimulant rather than a depleting plan of treatment. The first case which he gives is very remarkable, and is worthy of a brief abstract, as recent Anglo-American authors have not dwelt specially upon his symptom-group: A man, aged sixty-eight years, was suddenly seized with a fainting fit, which recurred several times in the day. For the three years before he was admitted to the Meath Hospital he had never been free from the attacks for any length of time, and had had at least fifty such seizures. A sudden exertion or a distended stomach was most apt to cause an attack. He had no convulsions, nor was there ever anything like paralysis. He was perfectly insensible for four or five minutes. The pulse was 28 per minute, and the arteries were "in a state of permanent distention, the temporal arteries ramifying under the scalp, just as they are seen in a well-injected subject." There was a soft *bruit* with the first sound. The threatenings of attacks he could recognize, and he had often warded off a seizure by turning on his hands and knees and keeping the head low.

In passing I may remark that you will find in this paper Stokes's original description of the Cheyne-Stokes breathing, which, though fuller, is not a whit better than Cheyne's account published thirty years before.

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* *Dublin Journal*, ix.

† Observations on some Cases of Permanently Slow Pulse. *Dublin Quarterly Journal*, 1846, p. 73.

Permanent slowness of the heart action and vertigo or syncope are the two distinguishing features of this syndrome. Do not forget that slowness of heart's action is the special feature, not simply a diminished number of pulse beats at the wrist. In myocarditis, in mitral-valve disease, and as an effect of digitalis the radial pulse may appear very slow—35 to 40 per minute—while the heart beats are exactly double. In the bigeminal type of heart beat the second pulse wave very often does not reach the wrist, and may lead a novice into the serious error of supposing that there is an extreme bradycardia. In "Adams's disease" the pulse rate may fall to 30 or 20 per minute, or, in extreme cases, even to 10 or 5.

The patients are usually advanced in years, and show often an extreme grade of arterio-sclerosis, the arteries feeling, as Stokes remarked, both full and hard.

The cerebral symptoms are those to which naturally chief attention has been paid. Vertigo is the most common, and is usually transient and oft repeated. Actual syncope of three or four minutes' duration, resembling closely the *syncope anginosa*, and doubtless of the same nature, has been the special feature in some cases; while in others the attack has been apoplectoid in its character, of longer duration, and has been complicated by convulsions. Huchard regards the slow pulse as a result of changes in the vagi centres, due to disease of the arteries of the medulla. He calls this form of arterio-sclerosis the *cardio-bulbar*. Transient disturbances in the cerebral circulation, so common in the subjects of advanced arterio-sclerosis, are responsible for the syncope and the apoplectoid attacks, which remind one of the temporary hemiplegias or monoplegias, the aphasias, and the transient paraplegias, to which these patients are subject.

Typical cases are not common. The most remarkable

instance recorded in this country is by Prentiss,* of Washington. A man, aged fifty-three years, with advanced arteriosclerosis, had for two years a pulse range of from 11 to 40 per minute. During this time he was subject to innumerable fainting spells, and had sensations of tightness across his chest. Before his death—which took place suddenly—he was delirious for several days. The heart was enlarged, but neither the aorta nor the coronary arteries were atheromatous. The sections of the medulla showed congestion of the vessels, but no apparent lesions. Dr. Prentiss presented this remarkable case at our meetings, and I remember very distinctly the advanced sclerosis of the arteries.

The following case belongs to this Adams-Stokes type, but the patient has, in addition to the vertigo and slow pulse, a sense of severe oppression in the chest, suggestive at least of an oncoming angina:

J. W., aged forty-six years, seen with Dr. Houston, of Troy, N. Y., February 13, 1895. The family history is good. With the exception of typhoid fever twenty years ago, he has always been well and strong. He has never had rheumatism, chorea, or syphilis; he has never worked very hard with his muscles. He is temperate.

His present symptoms began about two years ago with uneasy sensations in the chest on walking fast. At first not at all severe, within the past six months they have become very distressing. He has not been able to lie flat on his back, but can lie comfortably on either side with the head a little raised. He has no sharp pain in the chest, but uneasy sensations and a feeling of suffocation. He never has any cramplike or agonizing feeling; he has had at intervals severe vertigo, but has never lost consciousness. The unpleasant sensations in the chest are particularly apt to be present in the morning. He has had

* *Transactions of The Association of American Physicians*, vols. iv, v, and vi.

no dyspepsia, but of late has become very nervous about himself. He has to walk slowly, and on going up the slightest incline he feels the sense of oppression in the chest. He has no cough, no palpitation. About a year ago it was noticed that his pulse was very slow, and in the early morning it has on several occasions been counted as low as 20 per minute.

The patient was a healthy-looking man, with iron-gray hair; there was no arcus; the pulse was 34 to the minute; the tension was plus, the upstroke a little labored, and the pulse wave was well sustained. The chest was large; the cardiac impulse was not visible; there were no areas of abnormal pulsation; the aorta was not palpable in the sternal notch; there was no increase in the area of dullness; no apparent hypertrophy; no shock at the base; no thrill.

On auscultation, the sounds in the apex region were clear; over the body of the heart there was a roughish, systolic bruit, heard also at the aortic cartilage, transmitted feebly to the vessels of the neck. The second aortic was heard, but was not specially accentuated; there was no accentuation of the pulmonary second. There was no enlargement of the liver or spleen.

I heard from this patient in April, 1896. He has been a great deal better. He still has the brachycardia, but the vertigo is not so troublesome.

III. *Angina Pectoris sine dolore.*—The three elements in an attack—the pain, the sense of anguish, and the abrupt ending of life—may be dissociated. There may be only the severe pain, there may be a sense of *angor* and oppression without any pain—*angina sine dolore*—or death may occur without a moment's warning.

The recognition of a group of cases in which the element of *pain* is subsidiary was made by Professor Gairdner. His brief description is as follows:

"Apart from what has been variously termed cardiac asthma, dyspnœa, or orthopnœa, which in many cases receives its clear explanation from the associated states either of the pulmonary circulation or of the lungs, bronchi, and pleuræ, as disclosed by physical signs, there is often an element of subjective abnormal sensation present in cardiac diseases which, when it is not localized through the coincidence of pain, is a specially indefinable and indescribable sensation, almost always felt to be such by the patient himself. I make this remark deliberately, as the result of experience, and well knowing that it is liable to be brought into question in particular instances; that, in fact, a large part of what has been described under the titles given at the commencement of this paragraph has been inextricably confounded by systematic writers with the sensation, or group of sensations, to which I refer. To this group of sensations, when not distinctly accompanied by local pain, I have, in various instances, given the name of *angina sine dolore*, recognizing thereby what I believe to be its true diagnostic and pathological significance, and its alliance with the painful angina of Heberden; the pain in which, however, as we have already seen, is an exceedingly variable element, both in degree and in kind." *

Let me read you the histories of several cases of this variety:

Case XIII.—Mr. H., merchant, aged fifty years, who had suffered repeatedly from attacks of gout, consulted me on October 21, 1892, complaining of oppression and pain in the chest, and bronchitis. Throughout the summer he had had at times very severe pain in the region of the heart and down the left arm. When first seen he was anæmic, with a dilated heart and

* Reynolds's *System of Medicine*, art., Angina Pectoris.

an enlarged liver. With rest and iron he did very well. I saw him at intervals through the winter; the attacks of pain ceased, but he had severe cardiac asthma at night, which troubled him very much. I subsequently saw him in several attacks which followed the exertion of walking from the street car to my house, in which the feature of dyspnœa was subsidiary, and that of great oppression in the chest the most important. In these attacks the color changed, he became pale, looked very distressed and haggard, remained motionless, the forehead covered with sweat, the hands cold, the pulse feeble and irregular. After the attack he expressed himself as having had a feeling of indefinable distress without actual pain. There was no dyspnœa. The attacks at night were sometimes very severe, and he dreaded to go to sleep lest he should be roused in one. Though in the snmmer of 1892 he had had repeated attacks of what seemed to be true angina, yet he subsequently had only attacks of the kind just described.

In the spring of 1893 he became much worse; there were signs of dilatation of the heart, with the gallop rhythm, and a soft apex systolic murmur. He had cardiac dyspnœa, as well as attacks of severe oppression, and in one of these he turned on his side and died suddenly.

An attack of an *angina sine dolore* may be the very first indication of cardiac trouble.

An intimate friend, a man of about fifty-six years of age (Case V), of excellent habits and great energy, while on a visit to England, walking one Sunday afternoon with the late Dr. Hack Tuke up a slight acclivity, felt, as he expressed it, a sense of intolerable distress about the heart, turned pale, vomited, and for a few minutes could not move from the place at which he was attacked. He recognized the serious character of the paroxysm, and said that had there been the severe pain he would have called it angina. The attack was the starting point of a series of very distressing seizures, culminating in a protracted condition of cardiac dilatation, which kept him in his bed in Paris for several months. On his return he was

wonderfully better, took up his work, but soon had another breakdown, beginning with attacks of *angina sine dolore*. In one of these which I saw the pallor was extreme, the extremities were cold, a clammy perspiration bathed the forehead and face, the pulse was extremely feeble, and I thought any moment that he would die. After a protracted attack of cardiac dilatation, persistently feeble, irregular pulse, without any dropsy, but with the most remarkable psychical manifestations, he recovered, and was able for more than three years to attend to his duties. Then he had a sudden, more rapid breakdown, with cardiac dilatation, and he died between three and four years from the date of his first attack. I have already shown you sections from the coronary arteries in his case, which were sclerotic, and the myocardium was fibroid in places.

Case XXX.—E. H., aged fifty-four years, seen July 11, 1895, complaining of attacks of oppression in the chest, to which he had been subject for five years.

The patient was a remarkably healthy-looking man, of good color, of medium size, with iron-gray hair. Thirty years ago he had syphilis, but was thoroughly treated at Kreuznach, and he has had no troublesome symptoms. He married eight or ten years ago, and has healthy children. He has been a very heavy smoker from his eighteenth year; otherwise temperate; he has never done heavy work.

Five years ago he noticed that when making any extra exertion he had a sensation in the chest which compelled him to stop. After resting for a moment or two he could then go on. There was no pain with it. He was smoking excessively at the time, and after stopping the tobacco the attacks became less frequent; but for two years they troubled him a good deal.

Three years ago he retired from business and spent a year in Europe. When there he had his first severe attack. While going home after a hearty dinner with a friend, he was seized with a sensation in the chest, had to stop in the street, and was taken to his hotel. The feeling in the chest was as if everything in it was being drawn together and tightened, but without any sharp pain. He was very pale, he perspired, and the attack

lasted until the night. After the attack he had great depression of spirits.

The only other severe attack he has ever had was six weeks ago. He had been feeling very well, but before sitting down to dinner an annoying circumstance developed, and while still under the influence of the irritation he sat down and ate heartily. Immediately after dinner he had an attack of terrible oppression in the chest, feeling, as he expressed it, as though the life was being squeezed out of him. The slightest movement would increase the oppression. In the attack absolute quiet is what he desires. He does not even wish to be spoken to, but feels that the mind must be at rest. The immobility is evidently a very characteristic feature. When the sense of constriction and drawing is upon him, he says he could not force himself to budge an inch. In these severe attacks the pulse becomes very slow. The sensation is in the breast-bone in the middle.

In describing his sensations during a conversation of at least three quarters of an hour he did not use the word pain once, and states expressly that it isn't anything like pain, but an indescribable sensation of constriction and oppression. As he says, "he feels as if the end of everything had come"; at the same time "he feels so healthy that behind it, as it were, there is a feeling that he still has a long time to live."

In the two severe attacks a feeling extended into the muscles of the arms, not into the skin, he says, but there was a sense of strain and soreness in them.

The small attacks, as he calls them, recur with great frequency, and almost any day he has what he calls a *hindrance;* and if he makes any exertion of more than usual effort he has to stop short and wait a few moments until the sensation passes away. This may recur two or three times, and then, if he takes it slowly, he can subsequently walk two or three miles without any distress.

Two other circumstances which will bring on an attack are an unusually full meal and any mental worry. He never has the attacks at night.

The pulse was 72 when he was at rest; after his running

upstairs and down, 104; the tension was not increased; the superficial vessels were not sclerosed.

The apex-beat was only just visible in fifth interspace within the nipple line. The shock of the first sound was felt, not of the second. Area of superficial dullness was reduced by emphysema. Both sounds of the heart were clear; first a little flapping and valvular; no accentuation of aortic second sound. The examination of the heart was entirely negative. The liver was not enlarged.

July 12th. The patient stayed in town until I could see his condition in an attack. He had had two to-day, one quite light in the morning. He walked into the room somewhat deliberately, talked clearly and well, and had not changed in color. He said he had a sense of great distress just beneath the breast-bone. The pulse was small and hard, 103 a minute, with distinctly increased tension. After sitting down for a few moments his skin became moist, but he did not become pale. In the course of a few minutes the attack passed off with a feeling of glow. Afterward there was a very decided change noticeable in his pulse, which was softer and fuller, and of decidedly lower tension.

He was advised to stop smoking, and ordered a course of nitroglycerin. I heard from him in September and of him in May (1896). He still has the "smaller attacks," as he calls them.

The attacks may alternate with those in which agonizing pain is present, or they may entirely supplant the severer type. Some of the milder paroxysms, indicating the beginning, as it were, of the trouble, appear to be of this kind.

An iron-gray, healthy-looking man, aged sixty-four years, of good habits and excellent history, consulted me, May 25, 1895, about curious sensations in the chest. In October he noticed that when walking fast there was a peculiar sensation about the heart, as he said, "an aureole, which spread up his neck and head and went out to the hands." If he stopped for a moment, the sensation would "recede like a glow"; if he

went on, it would culminate in a pain which would compel him to stop. There was no sense of faintness, no dyspnœa, and he did not sweat. They have always followed exertion, and he has had as many as four or five attacks in a day. His arteries were a little stiff, but the aortic second sound was not accentuated. The top of the pinna of the left ear was calcified. He had never had gout. I heard from this patient on February 6th of this year. The attacks continue, though less frequent —only two or three a week. They are characterized by the same spreading glow, beginning at the heart, and lately the curious sensation has passed down the right arm alone.

IV. *Cardiac Asthma.*—Heberden insisted that in the paroxysm of true angina there was *no shortness of breath*, and yet we find a few years after his description the term *asthma* applied to the condition: *Asthma dolorificum* (Darwin), *A. arthriticum* (Schmidt), *A. convulsivum* (Elsner).

In reading the reports of the cases published within the first half century after Heberden's paper, it is very evident that much confusion existed, and nearly all forms of cardiac distress were termed angina. Desportes emphasized this on the title-page of his monograph (1811) on angina, which he said was a malady "presque toujours confondue avec asthma." The earliest and the latest, as it is the most urgent, symptom in heart disease is *dyspnœa*, which the older writers characterized as *asthma;* and as it forms a common feature in cases of angina pectoris it is not surprising that more or less confusion prevailed. Even Stokes does not seem to have had a very clear conception of the distinctions between these states, since he says that the disease which "most often gets the name of angina pectoris might be more properly designated as cardiac asthma."

What, then, is this condition? Go into the wards and ask the patients with valvular disease of the heart as to the very *first* symptom of their trouble. With scarcely an exception

they will answer, "Shortness of breath." Take a long series of histories of cases of arterio-sclerosis; you meet with the same complaint at the very outset. To the burly, obese draymen, to the heavy workers and the hard drinkers, and particularly if in addition they have been victims of the pox, Nemesis pays her first visit in an attack of shortness of breath—the *first indication of broken compensation* in an enlarged heart.

Clinically, we meet with various grades of intensity in this cardiac asthma. An exertion, the ascent of a pair of stairs, which may call forth only a fraction of the reserve force of a normal heart, may be too much for a right ventricle (in a case of mitral stenosis), or for a left ventricle (in a case of aortic insufficiency), and at the head of the stairs the patient pants, and is perhaps a little cyanosed. In chronic valvular disease such symptoms may recur on extra exertion for years without much significance; when the cardiac dyspnœa develops spontaneously, *without extra exertion*, the breakdown is not far off; and in the slow, too often watery progress to the grave no other symptom is so distressing to the patient. In cases of advanced arterio-sclerosis there are often attacks of dyspnœa of great intensity recurring in paroxysms, often nocturnal. The patient goes to bed feeling quite well, and in the early morning hours wakes in an attack which, in its abruptness of onset and general features, resembles asthma. There is usually a sensation of præcordial distress, a feeling of constriction and oppression, what the Germans call *Beklemmung*. Two other features about this form of attack will attract your attention—the evident effort in the breathing and the presence of a wheezing in the bronchial tubes and of moist râles at the bases of the lungs. The patient may spring from the bed and throw open the window in his terrible *air-hunger*, and he assumes an attitude most favor-

able to the working of all the accessory muscles of respiration. Slight cyanosis is usually present, and in severe paroxysms a cold sweat breaks out in the face and limbs. The pulse is feeble, often irregular, and very small, and on auscultation one hears either gallop rhythm or the fœtal type of heart beat. Death may occur in the attack, as in Dreschfeld's case, the history of which I gave you in Lecture III. This form of cardiac asthma occurs with great frequency in some of the subjects of angina pectoris, as in Cases V and XII.

And, lastly, the type of breathing known as *Cheyne-Stokes* is sometimes a form of cardiac asthma, and it is not uncommon in angina pectoris. The curious pause in his respiration of which John Hunter spoke was probably a period of apnœa in a paroxysm of Cheyne-Stokes breathing. It was first described by Cheyne in a case of fatty heart, and you will find it more frequently associated with chronic myocarditis than with any other form of heart disease.

The following case presents features of Gairdner's *angina sine dolore*, with characteristic cardiac asthma:

Mr. X., aged sixty-seven, seen March 9, 1895, with Dr. Claribel Cone, complaining of attacks of terrible oppression in the chest and a sense of impending death.

The patient was a very large-framed, well-nourished, vigorous-looking man. He had been always a very active business man, temperate, but a heavy smoker; he began in his eighteenth year, and has used as many as eight cigars a day.

For several years he has occasionally been roused from his sleep with a feeling of oppression in the chest, but it has never been very severe. For the past two or three weeks he has been very short of breath, and has had paroxysms of great oppression in the chest, with dyspnœa, feebleness of the circulation, cold, clammy sweat, and a sense of impending dissolution. During these attacks there is no actual pain. Mental excitement or muscular exertion will bring on the oppression and dyspnœa. He came to-day, however, in the street car, and walked half

a block without much difficulty. During the examination he had well-marked Cheyne-Stokes respiration.

The pulse was 104, of moderate tension, easily obliterated; the vessel wall was a little stiff. The apex-beat was feeble, just at the mamillary line; there was no shock of either sound to be felt; there was gallop rhythm at the apex, but no murmur. The second aortic sound was a little accentuated. The liver was not enlarged.

At 11 P. M. on the 9th the patient had a very severe attack of terrible oppression in the breath, with drenching cold sweat which soaked the clothing. During the attack the pulse was 104 and regular. The Cheyne-Stokes breathing became aggravated during the attack.

In this patient the attacks were more than ordinary cardiac dyspnœa. In addition to a sensation of awful oppression in the chest, there was a sense of impending death, and the cold, clammy skin showed profound involvement of the vaso-motor system.

For several weeks this patient seemed very ill. There were two interesting points in his treatment. The digitalis seemed to have helped him very much when the pulse tension was low; subsequently he got a great deal of relief from the full doses of nitroglycerin. Through the winter of 1895–'96 he remained pretty well, though subject to occasional attacks.

I can not leave this question of cardiac dyspnœa, of equal importance pathologically and clinically, without referring briefly to certain recent works upon it. A. Fraenkel, in the third edition of the *Real-Encyclopädie*, under Asthma, has a full and clear statement of the condition. Rosenbach's *Herzkrankheiten* has the most exhaustive discussion on the whole subject, full of suggestive ideas, but not easy reading, apparently not even to his countrymen, as Professor Martius speaks of the *Lehrbuch* as "in einer etwas dunklen Sprache geschriebene." The essence of Rosenbach's views on the relations of cardiac asthma and angina may be gathered from

the following sentence (page 377): "Dass nur eine Veränderung in der Art der Muskelcontraction, die die Stärke der Muskelleistung durchaus nicht zu beeinflussen braucht, wohl aber beeinflussen kann, Impulse für die sensiblen Bahnen liefert, die je nach der Erregbarkeit derselben und der Beschaffenheit der betroffenen Bahnen, die verschiedenen Formen von Schmerz und Angst auslösen, die wir als Stenocardie bezeichnen, während die Veränderung im Muskel die zur Leistungsschwäche führen, das Hauptsymptom des Asthma cardiacum, den wahren Luhthunger hervorruft." Or, as he says in another place, "die wahre Stenocardie ist ein blosses Zeichen der Regulationsstörung, das Asthma cardiacum ein Zeichen der Compensationsstörung."

Von Basch and his pupils * have endeavored to show that whenever, either from spasm or weakness of the left ventricle, the blood pressure in the auricle is raised, cardiac dyspnœa follows in association with two important changes in the lungs, viz., swelling and diminished elasticity = *Lungenschwellung* und *Lungenstarrheit.* The swelling, which may even be determined by percussion, results directly from the overdistention of the capillary network in the air cells, and to the same cause von Basch attributes the lessened elasticity. The ratio between the respiratory work and the intake of air is reduced; and, as Zerner remarks (*Studien*, Bd. iii), the peculiarity of cardiac dyspnœa is in this respiratory insufficiency, not in the rapidity and depth of the breathing. The "excursionsfähigkeit" of the lungs is lessened, and the amount of air inspired is not proportionate to the work done. Fraenkel also refers to the influence of the venous engorgement of the mucous membrane of the finer bronchioles as limiting the freedom of ingress and egress of the air to the alveoli.

* *Klinische und experimentelle Studien*, i, ii, and iii, particularly Bd. iii, 1896.

When we recall to mind the features of the attack in cardiac asthma and in certain anginal seizures, the similarity of the condition, as Huchard remarks, to an acute emphysema, the views of von Basch appear to possess at least a reasonable probability.

LECTURE V.

PSEUDO-ANGINA PECTORIS.

I. Neurotic group: (a) Hysterical and neurasthenic cases; (b) Angina pectoria vaso-motoria; (c) Reflex angina.—II. Toxic angina: forms of heart pain from tobacco.

An angina *notha*, false angina, was first described, so far as I can ascertain, by J. Latham in a paper (1812) on certain symptoms "usually, but not always, denoting angina pectoris." According to Huchard, the term pseudo-angina was introduced by Lartigue in 1846. Walshe called attention particularly to this condition, stating that "genuine angina pectoris is undoubtedly a very rare affection. On the other hand, I *almost daily* meet with a form of complaint combining, in a minor degree, many of the characters of angina; and to this imitation of the true disease I propose to give the name of pseudo-angina. I believe that herein lies the explanation of Laennec's notion (so discordant with the experience of English observers) that angina pectoris is of very frequent occurrence." The term which has come into general use, and is of no little value, has not passed without criticism. Balfour (*The Senile Heart*) says: "The term 'pseudo-angina' is often applied to anginous pains occurring before middle life, especially in the female sex, and yet we see that fatal angina may occur in one who is still but a girl. To talk of pseudo-angina is, however, a mark of ignorance rather than of refinement of diagnosis; for angina is but a symptom,

and if well marked, it should no more be stigmatized as 'pseudo,' because it occurs in youth, than the lesion with which it is sometimes associated should be called functional because it happens to be curable." And yet, not two pages off, he says: "But in what we may term—for want of a better expression—false angina, we have only to deal with the pain, the danger of which depends upon its cause," and in reality he subsequently acknowledges the wisdom of Walshe's division.

Burney Yeo says: "I do not admit a pseudo-angina of some authors. Hysterical imitative anginas, however, certainly occur. But . . . there is simply a gradation of severity and curability between the so-called cases of pseudo-angina and those of true angina."

Morison, too, in a recent paper,* questions the correctness of the term: "A case of true angina is one in which there is no doubt about the angina, and there is no mistake about the reality of the pain or breast-pang in many so-called functional cases." "The idea of spurious angina is only permissible in so far as the angina is not associated with demonstrable lesion," and tends to get well. Herein lies the essence of the whole matter—the symptoms, on the one hand, indicate the existence of a grave organic, usually incurable malady, and on the other, a condition very distressing, it is true, but rarely serious, and usually curable. The advantages in thus recognizing a functional group far outweigh any theoretical objections, and in a series of cases the forms are, with few exceptions, fairly well defined.

I have notes of some twenty cases of pseudo-angina pectoris, cases in which there were recurring paroxysms of severe, even agonizing cardiac pain, often with radiation. The ab-

* *Edinburgh Hospital Reports*, vol. iii.

sence of the mental element—*angor animi*—and the existence of collateral features, ætiological or symptomatic, served to separate them from the forms I have previously considered. I have only taken cases in which the seizures were paroxysmal and intense; if one counted all the slight cardiac disturbances in hysterical and neurasthenic patients as pseudo-angina, the list could be greatly extended.

At the outset let me emphasize three points of special significance:

First. Pseudo-angina is an affection of women. You remember that in the series of organic or coronary angina there was only one woman—a mitral-valve case—the only instance of true angina that I have seen in a female. Of the twenty cases of pseudo-angina, twelve were in women. If we exclude two cases of tobacco angina, there were only six instances in males. In my series it was much more common in married women; there were only two girls, each aged twenty-two. We can say then that pseudo-angina is almost as much a special disorder of women as true angina is of men.

Second. It occurs in younger persons. The average age of the subjects of spurious angina is much lower than in the other form—thirty-eight years in my series, the extremes being twenty-two and sixty years, the latter a woman who had had attacks for twenty or more years.

Third. The patients do not die. While fifteen of the subjects of true angina on my list are dead, every one of the twenty patients with pseudo-angina is alive; of several of the cases of true angina I have lost track.

Two main groups of functional angina may be recognized —the neurotic and the toxic.

I. Neurotic.—The cases in this group present a good many minor differences, either in the characters of the attack, or in the circumstances which favor its onset; and, based

upon these, various subdivisions have been made. A majority of the patients are either hysterical or neurasthenic, or the features of the attack are of themselves distinctive of hysteria. In others the vaso-motor phenomena are specially marked, while in a third set of cases the attacks appear to be excited reflexly, either by peripheral or visceral irritation.

(*a*) *Hysterical and Neurasthenic Cases.*—At the risk of wearying you, I will read the histories of a series of cases, from which you will get an idea of the varied features of the attacks. In every one of the first three cases the existence of genuine angina had been suspected, greatly to the distress of the patient or the relative. In the fourth the attacks were so anomalous that the presence of a tumor in the mediastinum was thought possible. An apprehension lest the true nature of the case should be overlooked is, naturally enough, the feeling uppermost in the mind of the attending physicians, whom to convince of the hopeful nature of the complaint I have sometimes found very difficult.

Case I.—Mrs. X., aged thirty-eight years, consulted me on January 9, 1894, complaining of attacks of agonizing pain in the chest.

Her father died at sixty-seven years, of cancer of the stomach; her mother, of nephritis, at the same age. There are no nervous affections in the family.

At the age of six she had rheumatic fever, and at twenty-one rheumatic sciatica, which has returned once or twice, but not within the past ten years. She has been married sixteen years and has two children. Until about six years ago she was subject at times to fainting fits, which would come on without any definite cause. She had them almost from her childhood, and I can not get either from herself or from her husband any accurate information as to her behavior in these attacks, which are apparently quite transient, and not associated with convulsive movement, nor does she apparently always lose con-

sciousness. She is a woman of a good deal of character and determination, and of executive skill, and does not give one the impression of being of a neurotic habit. She has always been able to do a great deal of walking and has not suffered specially with shortness of breath. Five years ago she had an attack of appendicitis, from which she recovered without operation. In the spring of 1893, when returning from Colorado, and after a good deal of mental worry, she had an attack of very severe pain in the chest. It came on after exertion and exposure to the wind in walking. The pain was of terrible severity, extended up the neck and down the arms; but she was able to move about in it and was a good deal excited. Since that attack she has been, at intervals, a little short of breath on exertion. She has had two of the severe attacks since; one last summer at the seaside, when walking on the sand, the other two months ago. Both of these were of great severity; the pain was agonizing; she became gray and cold and exhausted, and the skin was covered with a clammy perspiration. She states, too, that she had a sensation as though she could not live through it. One of these attacks was followed by a transient facial paralysis. Last August, for the first time in her life, she began to have headaches, which have recurred as often as two or three times a week. They are of the type of migraine, and come on with disturbance of vision; she sometimes sees figures and queer things; once she had hemianopia. The attacks prostrate her very much.

About the middle of December her feet and ankles began to swell, particularly at night. At first there was little or no pitting, but now they are sometimes swollen to the knees. The urine has been normal in quantity and without albumin or tube casts.

The patient was a well-nourished, healthy-looking woman; pulse 80, regular, without increase in tension, and the vessel was not sclerosed. The thyroid gland was not enlarged, and there was no puffiness of the face or above the clavicles. The feet and ankles at the time of examination were not œdematous. The examination of the heart and other organs was entirely negative.

A hopeful prognosis was given in the case, based on the view that the attacks, though severe, were probably pseudo-angina. The occurrence of migraine, with which pseudo-anginal attacks may alternate, and the swelling of the feet without evidence of heart or renal disease, were corroborative features. I have heard of this case several times; she got better, and the painful heart attacks, when I last saw her husband, six months ago, had not recurred.

Case II.—Mrs. F. R., aged forty-two years, seen April 9, 1894, complaining of attacks of agonizing pain in the chest.

The patient comes of a very nervous family, and one sister is in a lunatic asylum.

She was well and strong until two years ago; she has had five children, the youngest six years old. She has never had any special illnesses. She was not specially nervous as a young girl, and had no crying spells or hysterical attacks. Her domestic relations are congenial and satisfactory.

The present trouble began two years ago last February, when one morning she had an attack of severe pain in the chest. It began in the pit of the stomach, became most intense under the left breast, and extended round the shoulder. As she expressed it, she thought death had come. She got cold, broke out into a profuse perspiration, and during the attack was completely helpless. The attack lasted for about an hour and left her much prostrated. During the succeeding year she had about four attacks, each of great severity and identical in character. In the past year they have become more frequent; thus, she has had two in the past month. The last attack was on the 1st of April. She felt comfortable in the morning when she got up, but after breakfast felt a little drowsy and heavy, and lay down on the sofa. The attack came with the greatest rapidity and was so severe that she could not rise. The breath gets short; she feels a sensation of deathly coldness about the heart, and the chief pain is under the left breast. She can not move about, and when the pain is at its height she can not bear to be touched. As it comes on she loosens her clothing, but as the attack increases

in severity she is quite beside herself, tosses about, and is, as she says, almost dead. She groans a great deal, and in some of them has cried out very loudly. Her husband, who is a traveller, has only seen her in two attacks, both of which came on with great suddenness. She got very cold, the face became pinched and drawn, at first a little congested, and then pale. Twice she vomited in the attack. The duration has been from a quarter of an hour to an hour. She has had to have morphine hypodermically on several occasions. After they pass away she feels miserable and is wretched for two or three days. Sometimes the whole body is sore after an attack.

She knew of no special circumstance apt to induce an attack. She was low-spirited at times and noticed that she has been a little despondent prior to their onset. She has not been short of breath on going upstairs or uphill; not more than might be expected in a stout woman. Exertion has never brought on the attack.

The patient was a large, stout, healthy-looking woman. There was no arcus; the color was good; the tongue clean; the temporal arteries not sclerosed; pulse 84, readily compressed; the vessel wall was not sclerosed. There was slight throbbing in the vessels of the neck. Percussion was clear on manubrium. The cardiac dullness began on the fourth rib. At first right interspace there was a soft systolic murmur, and the aortic second sound was here a little accentuated. The first sound was loud and clear upon sternum; there was no murmur at the apex. There was no pain on firm pressure over manubrium or adjacent parts.

The soft bruit at the aortic area, and the accentuation of the second sound, made me a little suspicious of this case, though the general features of the attack were rather those of pseudo-angina. The subsequent history, as obtained from Dr. G. W. Norris and from her husband, on October 27, 1894, and July 7, 1895, shows that she steadily improved and the attacks have now ceased.

Case III.—Mrs. B., aged thirty-three years, seen with Dr. Smith, of Havre-de-Grace, February 14, 1895, complaining of attacks of pain about the heart and shortness of breath.

Her mother died of apoplexy at sixty; her father had a hemiplegic attack two years ago.

The patient was healthy as a child. At her seventeenth year had nervous prostration with headaches. She has never had any fevers, and has not had chorea or rheumatism. She has been married for twelve years; has had three children; the youngest is between three and four years of age.

The symptoms of which she now complains began about two years and a half ago. During her last pregnancy she had acute nephritis, but after delivery the dropsy disappeared rapidly. Within about six months she began to have attacks of palpitation and pains about the heart. These are very apt to come on five or six days before the menstrual period. She has two grades of attacks: In the severer type she gets very cold in the hands and feet. The heart begins to throb; she has choking sensations in the neck, and a sense of pain and oppression in the region of the heart. The pains do not extend to the arms. The face gets flushed, sometimes very much congested. She becomes very nervous, and the pain is so intense that she requires morphine. The attacks come on at any time, but exercise, heavy work of any sort, and worry, have seemed the most common exciting causes. In the milder attacks she has a little shortness of breath, the face becomes flushed, and there is a sense of oppression about the heart. They often pass off if she takes a hot drink or a dose of Hoffman's anodyne; she has never fainted. She has no dyspepsia, nor does she think that anything she eats ever brings on an attack. She has been exceedingly nervous and worried about her condition, particularly since a physician told her a year ago that she was liable to die suddenly. Up to a year ago she weighed only a hundred and fifteen pounds; she has rapidly increased in weight to a hundred and forty-three pounds. She was a healthy-looking woman of a florid complexion. She did not look of a nervous temperament. The pulse was good, 100 a minute, without increased tension; the arteries were not sclerotic. The condi-

tion of the heart was negative, the aortic second sound was ringing and accentuated. The pupils were equal; she had no arcus senilis.

There seemed very little doubt that this was a pseudo-angina, and I reassured her upon the question of sudden death. I heard of this patient on July 13th and on December 30th. She has not had a severe attack since February; for a few months she had "threatenings," as she calls them; since July she has been quite well.

The following case is of interest from the intensity of the paroxysms and the hyperæsthesia of the left arm. She had been alarmed, too, by the serious view which had been taken of her condition:

Case IV.—Miss C., aged twenty-two years, referred to me September 29, 1891, by Dr. Clark, of Skaneateles, complaining of remarkable attacks in the region of the heart.

The family history is good, and she has herself always enjoyed very good health. She is evidently a high-strung, nervous girl, who has studied hard. When quite young, about the twelfth year, she had for a time pain in the left side about the heart and sensations of coldness.

The present complaint has persisted for between two and three years. She describes a pain, more or less constant in the left front of the chest, which sometimes goes down the arm, which becomes numb. She says she is never without this pain, and that it sometimes keeps her from sleeping. Then she had sudden spells, in which she has a terrible sensation of spasm in the region of the heart, as though something had grasped her. It differs altogether from the other pain. In severe attacks it has lasted all night, and she has had to gasp for breath. She does not perspire. The left arm becomes numb, often tingles, and in severe attacks the numbness extends to the left leg. The left arm feels almost paralyzed and is tender, and she can not use it in the attacks. There may be headaches, but she is never sick at the stomach. She never has any special coldness of

the extremities. She has only had four of these very severe paroxysms within the year. During them she takes chloroform and nitrite of amyl. They have never been brought on by exertion, and she has been able to play tennis quite actively. Excitement and emotion most frequently cause them.

The patient was evidently very neurotic. She had no heart disease, no increased tension, and no sclerosis of the vessels. An interesting feature was the great sensitiveness of the left hand and arm. She jumped at once when I touched the wrist in order to feel the pulse. The various forms of sensation in it were perfectly normal. Though sensitive to the touch, she feels it numb and heavy. The sensitiveness did not extend to the skin of the chest.

The condition had been the cause of a good deal of alarm to her friends, and a diagnosis had been made by one of her physicians of a tumor pressing in the region of the heart. She was given a very favorable prognosis.

I saw this patient for a few moments about a year ago. She had entirely recovered from her attacks and, though nervous, seemed very well.

Hysterical angina in the male is usually a very well-characterized affection. The following cases are the most typical which I have seen:

Case V.—W. H., seen with Dr. Purvis, of Alexandria, aged thirty-two years, complaining of severe attacks of pain about the heart.

The patient comes of excellent German stock. His mother is alive and his brothers and sisters are well and strong; there are no special nervous troubles in the family. Though an hotel-keeper he has been very abstemious in the use of alcohol. He has never had syphilis. He has been nervous from boyhood. When about fifteen he had a fright, after which he had nervous spells, called fits, for several years. From his description, they were evidently severe hysterical attacks. At the age of twenty-three he had scarlet fever and diphtheria, and nearly lost his

life. For the past six years he has had a great deal of mental worry, and for nearly two years a good deal of extra financial strain. During this time he has had at intervals what he calls nervous attacks. He would get numb in his feet and then in his legs, and a sensation would rise in his body like a wave, making him cold and faint.

Dr. Purvis, who has seen him in the spells, says they are evidently hysterical. He does not lose consciousness.

For the past three months he has had different attacks, consisting of very agonizing pain about the heart, extending to the shoulders and down the arm even to the fingers, very frequently only to the index finger and thumb of the left hand. They have come on most frequently while walking. He catches his breath and has frequently had to sit down on a doorstep. He describes the pain as very agonizing, but he makes no mention of any sensation like that of impending death. His hands get cold; sometimes the feet are cold, and he has at times broken out into a profuse perspiration. The attacks have recurred with great frequency. He has had as many as four in the twenty-four hours. Worry, overexertion, and on several occasions a full meal, have caused attacks. They have increased rather than diminished during the past month.

The patient was a healthy-looking, well-nourished man, of good color, of fair physique, with black hair and eyes. The pulse was quiet (80 a minute), tension not increased. He flushed easily, and there was the most marked factitious urticaria and dermatographia. The apex-beat was not visible and not palpable. The superficial cardiac dullness was not increased. The sounds at the apex were clear. There was no accentuation of the aortic second, and there were no murmurs. There were no painful spots about the præcordia. The patient subsequently entered the private ward of the hospital, where he had several attacks of the character above described.

Case VI.—On May 23d I saw at the Rennert Hotel, Dr. R., aged thirty-three years, a physician from one of the Northern cities, who had had a series of most severe attacks dating from May 15th.

The patient, a man of very high-strung, nervous organiza-

tion, had had a very hard battle in life, overcoming almost insuperable physical difficulties. His general health had been very good. He had been a very hard student, and had done much work outside his ordinary professional duties. Three years ago, while engaged in instructing a class, he felt suddenly a terrible pain in the heart, and a numbness extended down the left arm and leg. He was unable to stand, but did not lose consciousness. He recovered from this attack in the course of an hour or so, and had no recurrence until the 15th of the present month. At 5.30 P. M., while in a cab, he was suddenly seized with an agonizing pain just below the left nipple. There were numbness and tingling in the left arm and leg. That night the pains recurred, and from his wife's account he evidently had a series of hysterical attacks; he became very emotional, wept, and had remarkable delusions. The pain was of such severity that he had to have morphine. The pulse was very variable, and at one time became extremely rapid, above 160. His face was flushed, not pale.

On Sunday, the 17th, he was better, and on Monday he was all right and attended to his practice. On Tuesday, while performing a minor operation, he had a recurrence of the agonizing pain. He said: "Words can not describe my torture, *but I went on and completed the operation.*"

On Tuesday evening he had another severe seizure, and had to have morphine hypodermically, and took chloral and bromide through the night.

On Wednesday he was in very bad condition, was nervous, emotional, and quite delirious. On Thursday he was annoyed by a cabman, and had an attack in the street, which upset him very much, but which was not, however, followed by delirium.

Altogether, in the past eight days, he has had five or six paroxysms of great intensity. In the attacks his wife says he is very restless, gets quite beside himself with the pain, and demands morphine at once. He has had all sorts of delusions, and has been in a most unnatural mental condition.

Patient was very healthy-looking, evidently very high-strung and nervous, a man who had for years lived far too intensely, and had worked very carelessly and with too much friction.

The physical examination was entirely negative. The pulse was quiet, without increase in tension. The heart sounds were clear, without accentuation of the aortic second. The vaso-motor system was extremely labile, and the slightest scratch was followed by an active reaction.

The persistence of pseudo-angina is sometimes very remarkable. In 1888 I was consulted, in Philadelphia, by an old friend, a physician from the Province of Quebec, who had very severe heart disease. While I was visiting him late one evening at the Lafayette Hotel, he asked me to step into the next room and see his wife, a woman sixty years of age, whom I found prostrate on the bed with her hands clasped over her heart, rocking herself from side to side, in an agony of pain. Her hands and feet were cold, the face somewhat flushed, the pulse small and rapid. I could not get an answer from her, but when I returned to the room the doctor said not to worry (I seemed anxious about her), that she would recover in a little while. He assured me that for more than thirty years she had been subject to these attacks, particularly when overanxious or worried. She was a very nervous woman, had been hysterical when young, and though at first her husband and other physicians thought the attacks very serious, they passed off so quickly, particularly under the influence of a hot whisky punch, that he had ceased to regard them as in any way dangerous.

(*b*) *Vaso-motor Angina.* — Vaso-motor phenomena are rarely absent in attacks of true angina, but they are even more pronounced in the nervous and hysterical subjects. Nothnagel has described a special type, angina pectoris *vaso-motoria.** In the four cases (all men) the symptoms consisted of peculiar sensations in the extremities or on one side

* *Deutsches Archiv. f. klin. Medicin*, Bd. iii, 1867.

of the body, with coldness and sometimes lividity of the hands and feet and sweating. With this there were palpitation of the heart, terrible præcordial anxiety or pain, and sometimes feelings of faintness. A striking feature in these cases was the tendency of the attacks to occur in the cold, or on washing the hands in cold water. Nothnagel regarded these vaso-motor phenomena as the primary features, and the cardiac embarrassment and distress as secondary to a widespread vaso-constrictor influence throughout the arterial system.

A good deal of discussion has taken place upon the propriety of recognizing this as a special type, and considering the frequency of vaso-motor changes in both organic and functional forms it does seem doubtful; and yet the cases are wonderfully well characterized and in the most pronounced degree always, I think, of the functional variety. In a large proportion the vaso-constrictor influences dominate, and there is pallor with coldness. I remember but one instance in which the vaso-dilator phenomena alone were marked.

In 1887 I saw (Case VII), in Toronto, a lady, aged thirty-five years, stout, well nourished, the mother of five or six children, who had been the subject, at intervals, of very puzzling and distressing attacks. Without any special reference to the menstrual period, and following particularly worry or excitement, she would experience a feeling of distress about the heart amounting to actual pain, and the vessels of the face and of the extremities would become congested, and she felt cold and numb. But much more distressing than these were the sensations of great pain in the back of the head and neck. The attacks would last for twenty-four hours or more, and were sometimes very alarming. I could not gather from her that the pains about the heart were ever of a very agonizing character, but they were always severe. I was asked to see her to determine

the presence or absence of a heart lesion. Both sounds seemed perfectly clear, and there were no signs of organic disease. I was much impressed with the neurotic condition of the patient, and suggested hysteria. I saw the patient in an attack, evidently hysterical; she was greatly prostrated, lay with the eyes closed, quite livid in the face, and the hands and feet were purplish in color and cold. She complained of great distress about the heart and agonizing pain in the back of the head and neck. The subsequent history has borne out the view taken of her case. Within a year or so she got perfectly well and has remained so, not having had an attack for nearly eight years.

Much more commonly there is pallor with the coldness. In women the attacks are apt to recur at or before the menstrual period.

Mrs. R (Case VIII), aged forty years, consulted me in 1890 about attacks of severe pain in the region of the heart, which had recurred at intervals for eight years, since the birth of her last child. They were particularly liable to come on during the menstrual period, or whenever she was subject to any special mental strain or worry. The pains were very severe, immediately under the left breast, and passed up the neck and down the left arm. She did not flush with them, but, on the contrary, got pale and felt very cold, particularly in the hands and feet, which sometimes sweated. The pains were not continuous, but recurred at intervals extending over a period of several days. Diet, she thinks, had no special influence. She slept badly and dreamed a great deal. The patient was a stout, well-nourished woman of good color; the pulse was regular, about 80; the arteries were not sclerotic. There was no heart disease.

In women, as you will have noticed in the reports I have read, the features of coldness of the extremities with numbness and pallor are very common. In men this type may occur in a most marked degree, and the diagnosis may be for some time in doubt.

Case IX.—A. B., aged forty-two years, seen December 30, 1895, complaining of paroxysmal attacks of terrible intensity, characterized by a feeling of suspended animation, as though the breath had left the body; at the same time the hands and feet get cold, and there is a sensation of stricture about the root of the neck.

The patient, who occupies a prominent position of trust, looks a healthy, vigorous man. His family history is excellent.

As a young man he was very well. He has never had syphilis; is a moderate drinker, and has used tobacco freely. Seven years ago, following a period in which he was very much overworked, he first had the attacks, which recurred for nearly eighteen months. At that time they caused him great alarm, but with the exception of two, they were not very severe. The present attacks date from just two months ago. He has been in his usual health, and knows of no special cause why they should have come on. A majority of the paroxysms have occurred at night, just as he was beginning to doze to sleep. He has had them also on the street, and seven years ago in one he had slight vertigo, and had to sit down on some steps, and he felt as he sat upon them as though they were rising and falling. The attacks may come on while he is sitting at his desk, or while he is reading quietly in his chair. Exercise is very apt to bring them on, and if he runs for a car or hurries upstairs he is apt either to have a severe attack or to experience a chilly feeling and the sensation which he constantly speaks of as though his breath had all left him.

The sensations which he describes in the attack are very curious. He lays special stress on the feeling that the respiration had ceased, and it gives him some relief to draw several deep breaths. With this is associated a sense of great stricture about the lower part of the neck, and a terrible sensation about the heart, as though it was his last minute. He feels strangely in the head, and thinks he has a very wild look. The face becomes pale, the hands and feet get cold as ice, and become very clammy with perspiration, and in several attacks he has had a feeling of numbness in the legs from the knees down. He lays

very great stress upon the sensation of coldness in the arms and legs, and says that on one occasion he took a warm bath, and even though the water was quite hot he still had a feeling of great coldness and numbness in his legs. In one attack the face and neck became very red and congested, and the nose bled profusely. There is invariably palpitation of the heart, and he has been told by his doctor that the pulse at the wrist becomes scarcely perceptible. In a paroxysm, seven years ago, he thinks he lost consciousness for a moment. He staggered and fell. In one attack at this time he had vertigo. As the paroxysm passes off he belches a great deal of wind. In several spells there has been a good deal of itching of the skin, and in one or two a marked twitching of the muscles. The duration of the entire paroxysm varies from two or three to ten or fifteen minutes. He finds that a strong drink of whisky will sometimes cut short an attack. In the two months since they recurred he has had on an average about four in a week. They have not all been severe. He has been much alarmed about them, and in several of the attacks both he and his wife have been greatly terrified.

Patient was a tall, well-grown, healthy-looking man. There was no arcus senilis; the pupils reacted readily to light. The pulse was soft and full, regular, tension low. The apex-beat was just within the nipple line, not forcible; slight throbbing in the vessels of the neck. The percussion note was everywhere clear; there was no increase in the area of heart dullness. The heart sounds were clear; the aortic second was not accentuated; the breath sounds were equal on both sides; there was no dullness in either interscapular region, and no bruit in the course of the descending aorta (a diagnosis of aneurysm had been made). The cervical glands were not enlarged. The examination of the abdominal organs was negative. The knee-jerks were normal. There was no Romberg's symptom, and the pupil reflexes were active.

January 1, 1896. The patient's wife came to-day to speak about her husband's condition. She says that last summer he had a few slight attacks. She mentions several features of interest, particularly the suddenness of the onset. For example,

he will awaken from a perfectly sound sleep in a most alarming paroxysm, and his hands and feet will become cold; the face is usually pale, and the heart will throb most forcibly. Within a minute or two his hands will become as wet as though they had been dipped in water. She remembers two or three attacks in which the face became quite congested and full instead of pale. He is greatly terrified, and always feels that he is going to die. What has reassured her always is the fact that within ten or twelve minutes, sometimes less, he is laughing and talking, quite free from pain. She does not think that he has been a very nervous man, and he has not had any special worries.

April 1, 1896. For the past two months this patient has been very much better, and, as he tells me, has almost recovered from his attacks.

June 1st. He has not had an attack for nearly four months.

(*c*) *Reflex Angina.*—And lastly, in addition to the purely hysterical and vaso-motor forms, there are cases in which the angina appears to be excited reflexly, either from peripheral or visceral irritation. You will find an interesting chapter in Huchard devoted to these reflex pseudo-anginas, and he has collected a number of cases from the literature. There are instances of anginous attack following a cervico-brachial neuralgia, of either traumatic or spontaneous origin. You remember in the histories of the cases of true angina how insistent many patients were as to the influence of diet. There is also a so-called gastro-intestinal form of pseudo-angina, in which attacks follow indigestion. The following is the only instance in my list in which the visceral irritation appeared to induce the paroxysms, or, to speak more correctly, in which the two conditions were associated:

Case XII.—Miss A., aged twenty-two years, seen April 4, 1893, complaining of severe attacks of pain in the region of the heart.

She belongs to a nervous family, and she has never been

very strong. She denies having had hysteria. Several times, as a child, she had slight rheumatism, and three years ago she was laid up with a more acute attack. Several members of her family have also had it. For years she has been subject to dyspepsia, particularly after eating too many sweet things. For a year or more she has had occasional attacks of pain in the chest, coming on particularly when she has indigestion. The pains are neuralgic in character, chiefly about the lower part of the chest, yet sometimes, to use her own expression, "they fly all over her." Lately she has been much alarmed by the occurrence of two attacks of great severity, the first about two months ago and the second a month ago. There was agonizing pain in the region of the heart with shortness of breath. Both were severe enough to require hypodermic injections of morphine. The pain, so far as she could localize it, was in the left side, in the region of the heart, not in the abdomen. On both occasions, though the severity of the pain was only, as she said, *for an hour or so*, yet for two or three days after she had more or less pain and distress. On both occasions she had dyspepsia, but she had not been specially nervous or run down. She does not know whether she got pale during the attacks, but she sweated after them. She takes a great deal of exercise, but has never had an attack brought on by exertion.

She looked a nervous girl and flushed easily. The examination was negative, with the exception of slight dilatation of the stomach.

II. Toxic Angina.—The second division of functional or pseudo-angina embraces cases due to the abuse of tea, coffee, and tobacco, substances harmless in themselves, but which if taken in excess may disturb the action of the heart. My experience with this form is extremely limited. In tea or coffee drinkers I have never seen attacks of cardiac pain which could be called angina; though paroxysms of severe palpitation, with distress about the heart and gasping respiration, are not uncommon in nervous women much addicted to tea. Tobacco,

as a rule, produces only slight and transient disturbance of the heart's action, but which may culminate in attacks of angina. When one considers how universal is the custom, the infrequency of severe heart symptoms in users of tobacco is remarkable. I pass months without seeing, in hospital or consultation work, an instance in which symptoms of any kind are due to it.

You all know, some of you have experienced, the acute toxic symptoms in beginning to use tobacco. The effects of the habitual use are very varied. To the large majority of persons the habit, in moderation, is harmless, to many it is beneficial. Among the injurious features those relating to the heart are perhaps the most important, certainly they are the most common. There are three groups of cases of so-called tobacco heart:

1. *The Irritable Heart of Smokers.*—Palpitation, irregularity, and rapid heart action are very common symptoms, particularly in young boys. They are often combined with dyspepsia; pain is not a special feature. There may be slight enlargement of the heart. It is a condition readily relieved by stopping the use of the weed. Disturbance of rhythm is the most constant effect of tobacco, and intermittence is more common than either slowing or hastening of the heart's action. Weakening of the vagus control is the more frequent, though in my own case the slightest excess in the use of tobacco causes intermission with slowing, not increase of the pulse-rate. An opposite effect—great rapidity with feebleness of impulse—is more common, and may develop suddenly in an habitual smoker.

2. *Heart Pain.*—Sharp shooting pains about the heart are not very uncommon in persons who smoke or chew too much. They may occur alone without disturbance of the cardiac rhythm or without the intensity and associated features of

an attack of tobacco angina. The following is a good illustrative case:

I. B., aged twenty-nine years, seen March 24, 1896, complaining of pain in the region of the heart just below the nipple. The first attack was four or five years ago, and it has recurred at intervals ever since. Shortly after the onset his physician suggested that it might be due to tobacco; and when he gave up smoking the attacks disappeared altogether. Since he resumed the habit they have recurred, and for the past year he has had them more frequently. The attacks occur at night, just after he has fallen asleep. He is awakened with a severe pain in the region of the heart, which almost takes his breath away, and makes him cry out at once. It rarely lasts more than a minute or two. The heart's action is not increased. He never has had any sweating and does not change in color, nor do his hands and feet become cold. He has never had any pain down the arm. It is always of the same character, sharp and stabbing, just below the nipple, and is intense enough to cause him to cry out. He has had as many as four or six attacks in the twenty-four hours. In the daytime the pain is not so severe, and the spells are more transient. He has never had an attack following exertion, and neither emotion nor errors in diet have any influence upon them.

He was a member of a very nervous family. He was himself a healthy, vigorous man. He had smoked from his boyhood three or four strong cigars, and when traveling, five or six cigars a day. He felt himself that the tobacco was responsible for the pain. He was a healthy-looking man, a little pale. The pulse was 76, regular, and without increased tension. The apex-beat was in normal situation; the heart sounds were everywhere clear. The second aortic was perhaps a little accentuated. There was no pain on pressure, and no hyperæsthesia.

He was advised to stop smoking altogether.

3. *Tobacco Angina.*—I have seen but two cases in which the severe paroxysms of cardiac pain appeared to be due to the abuse of tobacco.

Dr. ——, of ——, aged thirty-five years, consulted me April 13, 1891, complaining of severe pains in his chest and of numbness in the left arm. The patient has a very gouty history on both sides. He has been a hard-working practitioner, has been a moderate drinker, and has used tobacco to excess, both smoking and chewing. Four years ago, when he had been smoking very heavily, he had an attack of pain about the heart and down the arm, for which he consulted Dr. Pepper. He had very little trouble again until six or seven months ago, when the attacks recurred. He then consulted Dr. DaCosta, who said that he was gouty and without organic disease of the heart. Lately the attacks have been very severe, chiefly under the left margin of the sternum and reaching down the arm, which becomes numb and tingles. He has never had an attack in which there was a sense of impending dissolution. The patient was a healthy-looking man; the pulse was 78, the tension a little plus, but there was no sclerosis of the arteries. The examination was negative, with the exception that the aortic second sound was perhaps a little sharper and clearer than normal. He was told that he had no heart disease, and he was urged to live a temperate life, to give up tobacco, and ordered ten grains of iodide of potassium three times a day. After seeing him the first day I dictated the following note:

"In this case the gouty history and the accentuated second sound are perhaps suggestive of true angina. On the other hand he has been a very heavy smoker, is evidently nervous and worried about his condition, both of which factors must be taken into consideration."

I have seen this patient at intervals during the past five years. He lays very great stress upon tobacco as the cause of the attacks, and any indulgence is apt to be followed by severe pain. On February 17, 1894, in a letter he laid stress again upon the part played by tobacco; in a letter received recently he gives a very satisfactory account of himself, though he still smokes, and still has attacks. There is a feature in this case upon which Huchard lays a great deal of stress in tobacco an-

gina—namely, the occurrence of certain nocturnal spells almost like syncope. The patient states in the recent letter that "the strangest symptom of all is that just as I lose myself, and am about to drop to sleep, and often just after losing consciousness, I choke, sit up quickly, and feel for the moment as if 'the game was up.' There is no pain, no excitement of the heart, and yet this often occurs after having a choking fullness and distention in my throat, as if I was trying to force a great volume through a small space." He adds, "Sudden inhalations of tobacco smoke still give me pain about the heart, lasting for several minutes."

T. W., aged forty-five years, seen with Dr. Goldsborough June 12, 1895.

The patient was a very vigorous, healthy-looking man, and has enjoyed uniformly good health. In November, 1894, he had influenza and was wretched for two months after it. He had been a heavy smoker since his fourteenth year. Some years ago he gave up the habit for twelve months, as he had attacks in bed in which he felt as though the heart had stopped and he would have to jump out of bed and gasp for breath. He got well and has smoked heavily ever since.

On the 6th of January he had a sudden, severe paroxysm, to which he attributes his present nervous condition. He had been smoking on an average twelve strong cigars a day. The attack began with a peculiar feeling in the chest, not exactly pain, but great distress. He turned pale, belched gas constantly, perspired, was cold, could not lie down, and felt as though he was going to die. He had no agonizing pain, but he felt a sense of terrible oppression, and had numbness in both hands and wrists. The heart's action during this attack was scarcely perceptible, the pulse very feeble and fluttering. It lasted altogether two or three hours, and alarmed him very much. For several days afterward he felt prostrated and weak, and for a month he had a sort of faint feeling, particularly after eating. These faint attacks have distressed him very much. They would come on at intervals and he would turn pale and

sweat profusely. He never has actually fainted in them, but one day in the barber's chair he very nearly lost consciousness. They recurred for about two months after his severe attack. He has been very nervous and uneasy about himself, and has been greatly worried. He has stopped tobacco since January 6th.

The patient was a robust-looking man of good color. There was no arteriosclerosis. The apex-beat was within the nipple line, visible, readily felt, of normal intensity. There was no increase in the cardiac dullness. The heart sounds were clear, and there was no accentuation of aortic second. There was no enlargement of either liver or spleen.

Up to April 17, 1896, when last heard from, this patient had had no return of the attacks, and had been quite well.

As my experience of this form has been so limited, I will read you Huchard's statements as to the chief characters of tobacco angina:

1. "Angina pectoris due to tobacco assumes often the vaso-motor type (extreme pallor of the face with vertigo, contraction of the pulse, tendency to syncope, præcordial anxiety with or without pain, chilling of the extremities, cold sweats).

2. "The attack of angina is often associated with other manifestations of nicotine poisoning: vertigo, ringing in the ears, dysphagia, headache, a sense of suffocation and dyspnœa (nicotine asthma), sensations of general weakness, of cerebral confusion, of spinal hyperæsthesia, troubles of vision, etc. These symptoms may be dissociated from the paroxysms and observed separately.

3. "Those suffering from tobacco angina show, almost always, apart from or during the course of their attacks, disturbances in the heart's function: slowing with enfeeblement of the heart's beat, tachycardia or bradycardia, intermissions, arrhythmia, palpitation, tendency toward lipothy-

mia or syncope, attacks of palpitation with extreme irregularity of the heart (*folie cardiaque, delirium cordis*).

4. "The attacks of angina are often very painful and complete in their intensity and in the radiation of the pains. But it is in angina from tobacco that one sees particularly the imperfect and abortive forms, consisting of dyspnœa with slight præcordial anxiety, or simply of a little sense of uneasiness behind the sternum, with the sensation of stopping of the heart and the fear of impending death.

5. "Angina from tobacco shows generally spontaneous paroxysms; they may also be produced by exercise or exertion. It has then the clinical characters of angina from coronary artery disease.

6. "The paroxysms of *functional* tobacco angina due to spasmodic contractions of the coronaries disappear rapidly after the complete stopping of tobacco, a clinical feature common to almost all the symptoms of tobacco poisoning without lesions.

7. "This is not true of the paroxysms of *organic* tobacco angina due to organic contraction of the coronaries (through nicotine arterio-sclerosis). This form is more resistant; it disappears but slowly, or may be permanent; it is worthy of treatment with iodide of potassium.

8. "There exists another form of stenocardia, the most benign of all; it is due remotely but not immediately to nicotine; it follows dyspepsia produced by the abuse of tobacco; it is cured by the removal of tobacco and the disappearance of the dyspepsia."

LECTURE VI.

THEORIES OF ANGINA.

The importance of coronary artery disease.—Intermittent claudication.—State of the heart muscle in an attack.—Seat and cause of the pain.—Vaso-motor changes in angina.—Relations of spurious and true angina.

Coronary Artery Disease and Angina.—It would be impossible to discuss, even briefly, all of the theories which, from time to time, have been offered in explanation of this remarkable group of symptoms. Huchard has tabulated sixty-one opinions under six main theories! Under these circumstances it will be wise to start out with the statement of a generally accepted fact—viz., that in an immense proportion of all cases *angina pectoris vera is associated with disease of the coronary arteries and of the myocardium.* This, you will recall, was Jenner's original suggestion, which he enounced in the letter I read to you in Lecture I. Very shortly after the appearance of Heberden's paper the first reports of coronary-artery disease in angina were made by Fothergill—the great Fothergill, whose friendship with Rush and whose interest in the medical affairs of the American colonies endeared his name to the profession on this side of the Atlantic. In the first case which he reports there is no note upon the coronary arteries, but "on the outward muscular part (of the heart) near the apex was a small white spot as big as a sixpence, resembling a cicatrix," evidently a patch of fibroid myocarditis. In another case (which seems really

to have been one of *angina sine dolore*) the patient, who had a difficulty or incapacity to walk up a moderate ascent, died in a sudden transport of anger. John Hunter, who made the dissection, found "the two coronary arteries, from their origin to many of their ramifications upon the heart, were become one piece of bone." * The older reports, which corroborated the opinion of Jenner, are to be found in Parry's monograph; while the full statistics on the question have been collected with great pains by Huchard. In a supplementary chapter to his work you will find a summary of 145 autopsies in cases of angina, gathered from the literature. In 17 cases there was mention only of a lesion of the coronaries without further specification; of 128 there were 68 with lesions of both coronary arteries, 37 of the left vessel, 15 of the right, and in 12 the seat of the lesion was not stated. In the 128 cases obliteration or stenosis of the vessels had occurred, and of these in 121 there was atheromatous narrowing or thrombosis, in 5 embolism, and in 2 compression. Fatal cases are on record in which the coronary arteries have been found normal; most of these are instances of adherent pericardium or valvular disease. There are also fatal cases of tobacco and post-febrile angina in which the anatomical condition is stated to have been negative. Nothing is easier than to overlook myocardial changes, particularly in the older methods of examination, and a heart may present extensive fibroid disease with obliteration of arteries, which to the untrained eye looks healthy, or which may not show any coarse lesions of the aorta, or of the main branches of the coronary vessels. Or again, Krehl's method of serial section may show extensive myocarditis, with changes in the smaller arteries, in a heart apparently normal. Spasm of the coronary arteries

* *Medical Observations and Inquiries*, vol. v, 1774.

has been invoked to explain the sudden death in these cases, but it is much more likely that changes of a serious nature were overlooked (as from personal experience I know they often are) in the ordinary methods of examination. Ischæmia, a condition in which the heart muscle is imperfectly supplied with blood, is the main factor in all coronary lesions, whether narrowing of the orifices of the arteries, atheroma of their walls, or thrombosis or embolism of their channels.

In seeking to explain the relation of the arterial and myocardial changes to the symptoms of angina we pass at once into the region of speculation. On turning to the therapeutical indexes and finding a list of twenty or more drugs recommended in a given disease you may be quite safe in concluding that our knowledge of the treatment of the affection is, to say the least, imperfect; and so, when you read the tabular list of the theories of angina, covering nearly four pages of Huchard's *Traité*, you may feel assured that the last word has not yet been said upon the subject.

Theory of Intermittent Claudication.—The view which is based most directly on the coronary-artery disease is one which, as I shall tell you, dates really from the early part of the century, and finds its explanation in the remarkable phenomenon known as *intermittent claudication*. Bouley,* Sr., the celebrated French veterinarian, described an affection in the horse, in which, after being driven for fifteen or twenty minutes, the animal stops, the hind legs get stiff, and soon it is unable to stir. It may fall down, and apparently be in great suffering. In from half an hour to an hour it will recover and will go on comfortably for another fifteen minutes, and then an attack recurs. In such cases, post mortem, the artery of the affected limb has been found blocked

* *Nouveau dictionnaire pratique de médecine, de chirurgie et d'hygiène vétérinaires.* Tome deuxième, p. 540. Bouley and Renault, 1856.

with a clot, or, when both hind legs have been involved, the abdominal aorta has contained a thrombus.

Charcot, while an interne in the service of Rayer, described in man a condition corresponding to this intermittent claudication in the horse. He says * that one day a patient in the service told him that he was not able to walk for more than a quarter of an hour without being taken with cramps in the legs. After resting a while he would get better, and would be able to resume his walking, and then a crisis recurred. At the autopsy Charcot found a ball encysted in the neighborhood of the iliac artery, and a traumatic aneurysm which had obliterated the artery in its lower part. The circulation was carried on by collateral channels, which were ample to maintain the nutrition while the patient was quiet, and for a short period during exertion, but after a time, when the limbs were fatigued by the movements, the quantity of blood which reached them was insufficient, causing a relative ischæmia, with tingling, cramps, and impossibility of walking. He refers to the fact that the condition is often preliminary to gangrene, and narrates a case in which a patient with the affection had his leg amputated for gangrene. †

The credit of pointing out the analogy between this condition and angina pectoris, which is ascribed usually to Potain (1870), but which is maintained by Weber ‡ to be due to Brodie (1846), belongs in reality to Allan Burns, whose *Observations on Some of the Most Frequent and Important Diseases of the Heart* (1809) is a well-known storehouse of inter-

* *Leçons du Mardi.* Tome i, p. 45.

† Charcot seems to have felt hurt that his communication on so remarkable a phenomenon had not received any attention. He says: "Je n'ai pas encore rencontré, chose singulière, car mon mémoire de 1856, présenté à la Société de Biologie, n'est pourtant pas écrit en chinois, il me paraît écrit en français, presque en bon français, je n'ai pas rencontré, dis-je, un seul médecin qui ait tenu compte de mes observations."

‡ *American Journal of the Medical Sciences*, May, 1894.

esting facts. Since, so far as I know, this distinguished writer's connection with this supposed new theory has not been pointed out (except in the second edition of my *Practice*), I will read to you in full what he says on the subject: "Such a state of the arteries of the heart [referring to atheroma] must impair the function of that organ. It has been long known, that although the heart is always full of blood, yet it can not appropriate to its own wants a single particle of fluid contained in its cavities. On the contrary, like every other part, it has peculiar vessels set apart for its nourishment. In health, when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action the heart and every other part has its power augmented. If, however, we call into vigorous action a limb round which we have with a moderate degree of tightness applied a ligature, we find that then the member can only support its action for a very short time, for now its supply of energy and its expenditure do not balance each other; consequently, it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence. A heart, the coronary vessels of which are cartilaginous or ossified, is in nearly a similar condition; it can, like the limb begirt with a moderately tight ligature, discharge its functions so long as its action is moderate and equal. Increase, however, the action of the whole body, and along with the rest that of the heart, and you will soon see exemplified the truth of what has been said, with this difference, that as there is no interruption to the action of the cardiac nerves, the heart will be able to hold out a little longer than the limb.

"If a person walks fast, ascends a steep, or mounts a pair of stairs, the circulation in a state of health is hurried, and the heart is felt beating more frequently against the ribs than

usual. If, however, a person, with the nutrient arteries of the heart diseased in such a way as to impede the progress of the blood along them, attempt to do the same, he finds that the heart is sooner fatigued than the other parts are, which remain healthy. When, therefore, the coronary arteries are ossified, every agent capable of increasing the action of the heart, such as exercise, passion, and ardent spirits, must be a source of danger."

Burns discusses also whether the paroxysm was dependent on a state resembling paralysis, or on a spasmodic contraction of the fibres of the heart. He hardly thinks that the view of spasm is corroborated by any analogous facts in the animal economy. He says: "Do we ever, after the operation for aneurysm, see the muscles in a state of rigid action; or, when we apply the tourniquet only so tight as to impede the circulation, do we ever observe that the member is affected with spasm? In both cases we witness an induction of an extreme degree of debility, and we hear the person complaining of an unusual painful feeling in the limb, but still its muscles are in a state of inactivity. If these be the phenomena resulting from a deficiency of arterial blood in the muscular system in general, why should the heart be an exception? We know that this organ is principally composed of muscle, and we have therefore reason to believe that it is regulated by the same laws which govern other muscles."

I will read you this intermittent claudication theory as formulated by Potain, in 1870, and you will see how completely the distinguished clinician of *La Charité* has been anticipated by the old Glasgow professor: "If one considers the painful sensations, the disorders of the cardiac action, which constitute an attack of angina pectoris; if one but remembers that these paroxysms occur always after fatiguing movements, muscular efforts, or emotional disturbances—that

is to say, under conditions in which the heart is compelled to contract more frequently and to do more work; if one considers finally, that repeatedly some narrowing or ossification of the coronary arteries has been found in the bodies of victims of this disease; if one considers these facts, it will appear in every way probable that the heart does not escape from the common law, that it also becomes rapidly exhausted when its arteries can no longer give it the quantity of blood necessary for its increased activity; and that then it becomes the seat of painful disorders, just as in the case of the muscles of the lower extremity. Herein lies a principle which may be briefly expressed as follows: The symptoms caused by ischæmia become exaggerated whenever the diseased organ becomes active, because of the increased quantity of blood which this activity demands." *

It is easy to suppose that a narrowing of the orifices of the coronary arteries, or of the lumen of a main branch, can bring about conditions most favorable for the production of this intermittent claudication—*i. e.*, a state in which, so long as the heart is acting quietly, sufficient blood reaches its muscle; but if called upon to act more forcibly, by exertion or emotion, the larger supply, then needful to maintain the nutrition, might not be forthcoming, with the result of a relative ischæmia and disturbance of function.

What is the condition of the heart muscle in this ischæmia? Is it likely to be the same in the narrowing of atheroma and in the blocking from thrombosis and embolism? How shall we account for the remarkable disparity between the incidence of angina pectoris—a rare affection—and the incidence of coronary-artery disease—an everyday degeneration in persons above the age of sixty? With what special condi-

* Quoted by Huchard.

tions is the pain associated and what relation have the pains of pseudo-angina to those of the angina vera? What part do vaso-motor changes play in the process? These are among the questions which must be asked and answered before we can accept the intermittent claudication or, indeed, any other theory. We may discuss these points under three heads: the state of the heart muscle, the seat and cause of the pain, and the vaso-motor changes in the disease.

I. The State of the Heart Muscle.—During an attack the organ has been supposed to be either in spasm, or in a condition of paralysis, from imperfect blood supply or over-distention. Heberden, and many since, have regarded the heart in a paroxysm as in spasm or cramp; but Allan Burns, and after him Brodie (as quoted by Weber), urge against this view that the muscles in the condition following blocking of arteries are not in spasm, but rather the opposite; and, while not absolutely paralyzed, are, as Brodie says, in a state approaching to it. With this the clinical features of the attack are in accord, for although it has been noted in exceptional instances that the pulse beat has not been feeble or the cardiac rhythm disturbed, the general experience is that the left ventricle is weakened and the systemic arteries imperfectly filled.

The condition of the heart muscle in the attack is probably not always the same. For example, in a patient with ten or fifteen paroxysms daily we can not suppose that any serious organic change, as anæmic necrosis, develops in each attack. In such, as Allan Burns says, "the supply of energy and expenditure do not balance each other"; "a heart with the coronary arteries cartilaginous or ossified can discharge its functions so long as its action is moderate and equal, but if the circulation is hurried, the progress of the blood along the nutrient arteries of the heart is impeded and the heart be-

comes fatigued." A transient paresis from insufficient supply of oxygenated blood (and possibly, as has been suggested, from a sort of auto-intoxication with the products of imperfect metabolism) explains the cardiac weakness and the tendency to syncope, but affords not the slightest clew to an explanation of the main feature of the attack—the pain. Very different to this *relative ischæmia* of the cardiac muscle must be the condition following the blocking of a large branch by a thrombus or an embolus. The resulting anæmic infarct, if at all extensive, must cause not alone great weakness of the cardiac muscle, but at the site of the lesion the smooth uniformity of the waves of contraction must be seriously interrupted. This cardiomalacia may lead to rupture of the wall of the ventricle (eleven cases in Huchard's collection of autopsies) or may cause pericarditis. While the anæmic infarct is a well-recognized lesion in fatal cases of angina pectoris, it must be remembered that a paroxysm of pain is really a rare complication of this not infrequent change. It is interesting to note that the scars of infarcts have been found years after recovery from attacks of angina. Curschmann, in the discussion at the Congress f. innere Medicin, already referred to, mentioned two cases, one a man of seventy-five years, the other a woman of sixty, both of whom, some twenty years before death, had had severe attacks of angina from which they recovered with bradycardia. There were found old fibroid changes in the myocardium with obliteration of branches of the left coronary arteries. We may say, then, that the evidence, such as it is, favors the view that the heart muscle in the attack is in a state of paresis. This, however, may not be general; it may be confined to the left ventricle or to a part of its wall; but weakness in itself offers not the slightest clew to the cause of the pain.

The view of Heberden that the heart muscle during the

attack is in a state of spasm has been supported by many writers, notably by Latham. The existence of spasm of the heart during life can not be inferred from the empty and contracted condition of the left ventricle post mortem. Relative ischæmia in the territory of one coronary artery or of its main branch, still more an area of anæmic necrosis, might readily bring about conditions favoring cramp, not necessarily in the affected region (very unlikely, indeed, in an infarct), but in contiguous muscular districts, the rhythm of whose motion would be interrupted and disturbed. I do not know of cramp in the voluntary muscles produced under analogous conditions, but I may remind you of the horribly painful cramps in the legs in the exhaustion following the prolonged use of untrained muscles, and the cramps in the calves and feet in chronic arterio-sclerosis. Pain, the special feature of the angina attack, is explained by the cramp theory. The most intense suffering which can be experienced is associated with muscular contractions of the tubular structures, as in intestinal, biliary, and renal colic, and in the contractions of the uterus in parturition. And observe that this agonizing pain is in parts not endowed, so far as we know, with very acute sensibility. Theoretically there is much in favor of the idea that in the most powerful muscular organ of the body irregular cramplike contractions, even if localized, might be accompanied by painful sensations, which could attain the maximum intensity present in an angina attack. But this brings us directly to a discussion of

II. The Seat and Cause of the Pain in Angina.—There is one inexplicable feature which baffles all suggestion, and gives us pause in an uneasy apprehension lest we should know even less than we suppose. I refer to the extraordinary variability in the incidence of attacks in cardio-vascular lesions apparently most favorable. Why should true angina pectoris

be so rare in hospital patients, and so rare in women? There must be some peculiar state of the nervous system, some undue susceptibility, as Sir Richard Quain says, which, in the presence of certain conditions, is really the essential factor. Like epilepsy, to which it has been compared by Trousseau, and more recently by B. W. Richardson, we know the signs and symptoms and can give a dull catalogue of predisposing causes, but of the intimate cause we know nothing, and can only formulate our knowledge in general statements, such as have been given in the lecture upon ætiology.

The seat of the pain is undoubtedly in the heart itself. The irradiation is a remarkable phenomenon to which we have no other exact counterpart in visceral disease—none, at any rate, which is so pronounced a feature. Anstie, Allen Sturge, and others have suggested the possibility of a central origin of the whole trouble; and I would here remind you of the interesting observation of Eichhorst that atrophy of the muscles of the ulnar side of the left hand may follow repeated attacks, which would suggest central changes in the spinal cord. Which set of nerves in the heart is chiefly involved, and what part the intrinsic ganglia play, we do not know. It has been suggested by Sansom that the pain is due to involvement of the sympathetic fibres, the feeling of impending death to the influence of the vagi; which recalls to mind Laennec's opinion that when the pain was in the heart and lungs the vagi were affected, and, when there was simply a sense of stricture of the heart without difficulty of breathing, the grand sympathetic was involved. Four possible explanations of the pain may be mentioned:

(*a*) *Cramp of the Heart Muscle.*—In discussing the state of the heart during an attack I have spoken of this view, which has much in its favor, particularly in cases with anæmic infarct, but it seems scarcely applicable to all—for instance, to

the cases with frequently recurring attacks, in which one can not possibly suppose infarcts to be present, though the scars, of course, persist. The analogy with painful spasm in other hollow organs, usually very insensitive, is also suggestive. That Heberden and Latham—still masters in Israel—stand sponsors for this view, and that so acute a modern observer as Rosenbach, should conclude that the pain is due to "changes in the form of muscular contraction," commend it strongly to our consideration.

(*b*) *Distention and Stretching of the Cardiac Walls.*—Traube held * that the symptom-complex of angina pectoris resulted from a rapidly increasing distention of the walls of the ventricle, which, in consequence of defective nutrition, were more yielding. When, owing to increased pressure in the aortic system, this distention became excessive, the nerve elements in the heart wall became stretched and bruised, causing the pain. You will find a very careful elaboration of this theory by Lauder Brunton in the *Practitioner*, vol. xlviii. A paragraph in a lecture by T. K. Chambers also suggests this idea: "The pain has the same tearing and paroxysmal character that you find accompanying the distention of hollow fibrous organs, usually insensitive, such as the stomach, the colon, and the bladder. The pain is of the same nature as that felt in overstrained tendons or muscles wearied out by sustained effort; it appears associated with the stretching of usually insensitive fibres, and is sometimes the most dreadful agony the body can bear, as the inventors of racks and other instruments of torture well knew." † Of course, this is a possible explanation, but it raises a problem insoluble as the original one—why, if extreme dilatation is a cause, angina does not occur more often. There must be surely some addi-

* *Gesammelte Beiträge*, Bd. iii, p. 183.

† *Lectures, chiefly Clinical*, fourth edition, p. 315.

tional factor, or attacks would be of everyday occurrence. The relation of angeiospasm to the attacks will be discussed later.

(*c*) *That the Pain is in the Arteries.*—Allan Burns spoke of the pain following the tying of an artery and the application of a tourniquet. Sensory nerve endings have been demonstrated in the arterial walls, and it has been suggested frequently, in recent discussions on angina pectoris, that the main element of the attack may be *vessel pain*, due to either angeiospasm or thrombosis. There may be—there is not always—great pain in the blocking of a large vessel, artery, or vein by a thrombus or embolus. The name phlegmasia alba *dolens* emphasizes a prominent character in the plugging of the femoral vein, and, as I have just said, the pain after ligation of the femoral or the application of the tourniquet is often very intense. Nothnagel refers also to the pain in the head in blocking of large cerebral vessels. It is not unreasonable to suppose that pain of the same nature may occur in blocking of the coronary arteries, though I do not call to mind the existence of special pain in embolism or thrombosis of arteries of the size of the coronary vessels in other organs. Moreover, as I have already said, we can not suppose that in each attack a thrombus develops. Angeiospasm is a much more likely cause of the pain, and it may be associated in some cases with blocking of a vessel. There are the analogous conditions of migraine with its vascular spasm and intense pain, and the vascular changes with pain in Raynaud's disease. Balfour has an interesting paragraph upon this question of pain in the arteries:

"That ischæmia does give rise to pain, even of the most atrocious character, is sufficiently attested by the agony that attends compression of an artery for aneurysm, especially at the moment the vessel becomes completely occluded; the

pains, arising from a similar cause, that precede the appearance of gangrenous patches in a limb affected with senile gangrene; and those which precede, accompany, and follow attacks of local asphyxia (Raynaud's disease). There is every reason to suppose that the arterial spasm, which is so evidently the cause of local asphyxia, and which takes so prominent a share in the production of an attack of angina vasomotoria, occasionally invades the heart, either as part of a general condition or, it may be, as a distinctly local affection, and that this is a very possible cause of those anginal attacks where no other seems obvious" (*The Senile Heart*).

(*d*) *That the Pain is a Neuralgia, either Functional or due to a Neuritis.*—This most widely held view regards angina pectoris as a form of neuralgia or neuritis affecting the nerves of the heart. Huchard mentions twenty-two modifications of this theory, which dates from the early part of the century, when, in 1808, Baumes ranked the disease as a retrosternal neuralgia (sternalgia). Laennec gave it his strong support and held that either the pneumogastric or sympathetic division of the cardiac nerves might be implicated, and with either of them the brachial plexus. Corrigan, Romberg, Bamberger, and others held the same opinion. Then in 1863 came the observations of Lancereaux on changes in the cardiac nerves and ganglia, which were confirmed by Peter and others. Huchard states (second edition, 1893) that there were only twelve observations on neuritis of the cardiac nerves, of which six were associated with disease of the coronary arteries. More recent literature, so far as I know, does not furnish additional cases, and the whole question of minute histological changes in the sympathetic nerves and ganglia in various disorders must be reviewed with the help of the new technique.

Against this theory may be urged the common observa-

tion that the cardiac nerves may be seriously implicated in aneurysm, in mediastinal tumors, in adherent pericardium, and in the exudate of acute pericarditis without causing the slightest pain.

Again, in the attack of angina, though the pain is a prominent feature, it is a part, and in a severe attack the minor part, of the paroxysm. The *angor animi* is very unlike anything met with in neuralgic affections. Moreover, the mode of onset following exertion or emotion is not a feature of neuralgia, and this view affords no solution of the sudden death which sometimes follows. In its paroxysmal character and radiation, and in its intensity, the pain is much more like that of biliary and renal colic; with the latter, indeed, I have heard a patient who had experienced both compare it.

Of course, the pain suffered in an attack of angina is a manifestation of disturbed function of the nerves. Such disturbance, when associated with pain, may be called neuralgic, but it is evident, from what has been stated, that there is something in addition, which puts the attack out of the category of ordinary painful affections of the nerves. There are many conditions about the heart in which the nerves are directly implicated with which neuralgia occurs. I have already told you that there is no constancy in this, and there may be old pericardial adhesions, fresh epicarditis with direct involvement of the superficial nerves, or there may be sclerosis of the root of the aorta, aneurysm or tumor with pressure on the pneumogastric, without any pain whatever. But again, in all of these conditions there may be recurring attacks of pain about the heart, sometimes of great intensity, and even simulating that of true angina.

III. Vaso-motor Changes in Angina.—In Lecture III I mentioned the striking vaso-motor phenomena of the attack —the pallor, the coldness, and the sweating—and in the last

lecture I spoke of a special type of pseudo-angina in which these features dominated the scene. They play a conspicuous rôle both in the functional and organic forms. Naturally, one approaches a vaso-motor problem with a good deal of caution, since it lends itself with singular aptness to theoretical vagaries and to all kinds of speculation. It is well to remember that, as Foster remarks, the vaso-motor nerves are servants, not masters, in the matter of regulating the calibre of vessels and altering the blood pressure.

I have already spoken of the state of the arteries during the paroxysm, and have given a summary of my personal experience on this moot question. The general opinion is that in true angina there is an early angeiospasm with great increase in the blood pressure. Sphygmographic tracings during the attack have not often been made. Lauder Brunton's observation is particularly interesting, and I show you here the tracings which he gives, taken from the radial pulse before and during an attack. It can not be doubted, I think, that in many cases an important factor is, as Mitchell Bruce expresses it, too much pressure ahead of the driving power; but this widespread peripheral spasm is probably a secondary phenomenon, excited reflexly through influences on the vaso-motor centre coming from the heart itself or from other parts. Morison, of whose paper in volume iii of the *Edinburgh Hospital Reports* I have already spoken, gives the notes of a patient with aortic insufficiency, in whom during an attack the pulse tension was low, and he thinks that even in the organic form there may be considerable variations, more especially in the cases with or without insufficiency of the aortic valve. It may be questioned, indeed, whether the tracing in Brunton's case really represents a great increase in the tension, or whether it does not mean that the left ventricle was in a condition of feebleness or dilatation, and the pulse tension extremely low.

I show you here by way of contrast the tracings given by Huchard, in which, as you notice, the one in the interval between the attack with the low tension resembles very much that of Brunton's during the attack with supposed high tension.

A majority of patients with true angina have reached an age in which naturally the blood tension is increased, and in almost every instance the exciting causes of the paroxysm are those which raise the arterial pressure—mental emotion, muscular exertion, cold to the periphery, and dilatation of the stomach. You will find in Brunton's paper an admirable discussion of the importance of these factors in raising the blood pressure, and in bringing about the anginal paroxysm.

Favoring, too, this view of widespread angeiospasm is the circumstance that in certain cases of Raynaud's disease angina pectoris of a very severe type has occurred, and has even proved fatal. The most interesting case of this kind in the literature is reported by Richard Cleeman.* A man, aged sixty-two years, had from his fiftieth year severe attacks of Raynaud's disease, chiefly in the hands, which occurred usually in the winter season. One day he had an attack of agonizing substernal pain lasting for two hours, and of such intensity that he was greatly prostrated. The pains radiated down both arms. During a period immediately preceding this he had had very pronounced attacks of local asphyxia and local syncope, chiefly in the hands. A week later he was found dead in bed.

The association of migraine with angina pectoris, particularly the vaso-motor form, has been long recognized, and in two cases in my series the subjects had been great sufferers with this disease.

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* *Transaction of the College of Physicians,* Philadelphia, 1892.

There are two possible explanations of the vaso-motor phenomena of angina pectoris. In the first place a widespread vaso-motor spasm, excited reflexly by cold, by emotion, by flatulence, etc., throws a great strain upon the left ventricle with, as Traube thought, distention, stretching, and consequent pain. Of course, in this widespread angeiospasm the coronary vessels themselves may participate, and it is not at all improbable that in the hysterical and vaso-motor varieties of angina the entire symptom-complex may be vaso-motor. The possibility of a local (coronary) angeiospasm may be admitted in the toxic cases and in organic disease of the coronary arteries. On the other hand, the widespread constrictor influence in the systemic arteries in an attack of true angina may itself be a vaso-motor reflex. Hegar showed experimentally that a great increase in the general blood pressure could be excited reflexly on the injection of nitrate of silver into the peripheral artery of a rabbit. In the same way the pallor, coldness, sweating, and general vaso-contrictor influences in true angina may be excited reflexly by afferent impulses from the coronary vessels themselves.

Relations of Pseudo to True Angina.—One other matter remains for brief comment. What relation do the phenomena of spurious angina bear to those of the organic affection? Huchard insists upon the absolute separation of the organic form associated with coronary-artery disease from the various other types of cardiac pain. He says: "*Il n'y a pas plusieurs angines de poitrine; il n'y en a qu'une seule, l'angine coronarienne.*" According to him the pseudo-anginas are neuralgias of the cardiac plexus due to various causes, or a vaso-motor neurosis. It must be acknowledged that the attacks of vaso-motor angina and of the form seen in nervous and hysterical women have many of the characters of a paroxysmal neurosis, resembling indeed in certain particulars mi-

graine. Closely allied as no doubt many of the underlying conditions are, and simulating often the features of the genuine attack, I fully concur with Huchard and others who separate the functional from the organic form; and, while the former come very properly in the category of paroxysmal neuroses, the true angina presents features entirely unlike a neuralgia. The chief objections have been well and clearly put by Fagge: "But for a neuralgia to prove habitually fatal is without precedent. Moreover, angina pectoris differs from all neuroses in being generally, if not always, associated with the existence of organic lesions in the heart or in the great vessels, although it would seem that no one lesion is constantly present; this, at any rate, is true of the cases that destroy life. Thirdly, it is unlike a neuralgia to attack, as angina does by a large preponderance, more males than females—as many as ten men to one women."

In the neurotic form the fundamental error appears to be a vaso-motor instability, for which S. Solis-Cohen has suggested the name vaso-motor ataxia, a term which really defines the condition, a loss of the power nicely to balance the distribution of blood in the vascular territories. In the organic form not only is the question much more complicated, but there are features quite inexplicable in the present state of our knowledge—notably the haphazard incidence in anatomical conditions apparently identical, the causation of the pain, and the relation of the blood pressure, cardiac, coronary, and systemic, to the phenomena of the attack.

Were the problems of blood pressure solved, angina pectoris would be an open book to us; but in spite of the unceasing work of the past thirty years much obscurity remains, with not a little dissonance and discord. The trained student among you who wishes to get upon a working basis should

study the articles of Porter referred to in Lecture I, the important monograph of Roy and Adami,* the *Heart Studies* † of Ewart, von Basch's works and his recent brochure on *Gefässstarre* (Angiorhigosis) ‡—these, with Tigerstedt # as a sort of Baedeker, will promote his enlightenment. The less ambitious will be content with the lucid account in Stewart's *Physiology, facile princeps* among manuals on the subject.

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* *Philosophical Transactions*, 1892.
† London, 1894.
‡ Vienna, 1896.
# *Lehrbuch der Physiologie des Kreislaufes*, Leipsic, 1893.

LECTURE VII.

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF ANGINA.

Anomalous cases of heart pain.—Elements in the diagnosis of true angina.—Differentiation of true and pseudo angina.—Prognosis.—Treatment of angina pectoris vera.—Treatment of false angina.—Conclusion.

Diagnosis.—One must be a professional Ulysses in craft and wisdom not sometimes to err in estimating the nature of an attack of severe heart pain. There is no group of cases so calculated to keep one in a condition of wholesome humility. When you jostle against a hale, vigorous specimen of humanity, who claps you on the back and says, "The deuce take you doctors! I have scarcely yet got over my fright," you would like to forget that five years before you had almost signed his death warrant in a very positive diagnosis of angina pectoris vera. On the other hand, Mr. X. has left you with the full assurance that his cardiac pains are due to overwork or tobacco, and you have comforted his wife and lifted a weight of sorrow from both by your most favorable prognosis. With what sort of appetite can you eat your breakfast when, a week later, you read in the morning paper the announcement of his sudden death in the railway station? Or take another aspect—poor Mrs. Doe has gone softly all these years in the bitterness of her soul since you took that grave view of her vaso-motor or hysterical angina!

As a rule you will have little or no doubt as to the existence of angina. The chief difficulty is in deciding upon the

functional or organic nature of the trouble. There are, however, extraordinary cases of recurring pain about the heart, of terrible severity, the nature of which may be very obscure. The following is one of the most remarkable cases of this kind which I have met:

J. H. McC., aged forty-nine years, seen April 28, 1887, complaining of attacks of terrible substernal pain. He was a large, active man, weighing a hundred and ninety-five pounds. With the exception of a chancre at his fifteenth year, which was followed by an ulcer on the leg eight months afterward, and typhoid fever three years ago, his general health has been good.

Twelve years ago (1875) he consulted Dr. Weir Mitchell for the following symptoms: Every morning, about one o'clock, he was aroused from sleep with severe pains in the lower part of the chest, beneath the sternum. At first the attacks occurred every night, and then at intervals of four or five days. The pain was not like a cramp or a spasm, but dull and severe, intense enough to make him get up and walk the room. For nearly ten years the attacks made his life a burden. They ceased abruptly in 1885, since which date he has been quite well until March 28th of this year. He now has the attack every day about 1 A. M. He goes to bed at ten o'clock and falls to sleep comfortably. Usually about 1 A. M., sometimes at three or four o'clock, he is aroused by a fixed pain beneath the sternum between the fourth and fifth costal cartilages. It is never transmitted down the arm; he is never doubled up with it, nor does he turn pale or sweat. He occasionally belches wind during the attack. The pain at times is so severe that he has to take an anæsthetic. Thus on the 29th he awoke at 3.30 A. M. in terrible agony, and had to take ether. He slept until after five o'clock, when the pain again came on and persisted until noon. He says he has noticed that it is often worse after the rest of Sunday. The examination was entirely negative. The arteries were not stiff; the heart's action was regular; the aortic second sound was not accentuated. He winced a little on deep pressure just above the ensiform cartilage. The urine was normal.

He had taken iodide of potassium previously without, he

thought, any benefit. Considering, however, the history of syphilis, I insisted that he should take it again. I heard from him on several occasions for a year or so, and there was not very much change in his condition. I have since lost track of him.

A somewhat similar case to this is reported by Dr. Randall, of Decatur, in the *Medical News* for March 11, 1893. The patient was a healthy man who had remarkable seizures of agonizing pain in an area twelve inches in diameter over the heart, which recurred nightly from August 1st to December 16th unless he remained awake and out of bed. The patient was a self-possessed, unimaginative man, and had never had similar attacks. The pain was sometimes greatest in the epigastrium, sometimes in the præcordium, and with it there were tingling and numbness in the arms, and a sense of suffocation. There was no dyspnœa or asthmatic breathing. The patient could avert the attack every night by vigilance. It is difficult to decide upon the nature of such cases.

The important elements in diagnosis are the sex, the presence of cardio-vascular disease, and the phenomena of the attack.

Upon the infrequency of true angina in women I have already dwelt, and you must be cautious even when the general features of the case are most suggestive. Such an instance, for example, as the following should not deceive you:

Mrs. R., aged forty-two years, seen October 25, 1894. She has always been a healthy woman, but has had much trouble and worry. Her husband had attacks of angina pectoris, and died a year ago of heart disease. Her mother died in an attack of angina pectoris three years ago. For nearly a year she had been unable to rest comfortably on her left side, and had been much troubled with pains about the heart, which were sometimes of great severity, and were then accompanied by a feeling of numbness in the left arm extending to the fingers.

Neither diet nor exercise seemed to make any difference, and she never had dyspnœa. Sometimes the whole of the left side, including the neck, would feel stiff and sore. Though she had been free for a few days from the sensations of pain, she had not within the past year had a week of complete intermission. She had evidently worried very much about it, and dreaded consulting a physician, lest she should be told she had the disease of which her mother and husband had died.

The patient was a healthy looking woman. The pulse was quiet, without increase in the tension, and the arteries were not stiff. There were no signs of hypertrophy or dilatation of the heart; the sounds were normal. The urine had a specific gravity of 1.018, and was free from albumin and tube casts. I have seen this patient on several occasions during the past two years, and her condition has improved very much.

There are cases, too, in which hysterical angina occurs in women with aortic valve disease. The only instance of the kind which has come under my notice is the following:

Mrs. K., aged thirty-eight years, seen February 24, 1890. The patient was an unusually bright, able woman, who had for several years lived under a serious mental strain. She had severe rheumatism as a child, and she had been told by several physicians—among others the late Dr. Austin Flint and Dr. Da Costa—that she had heart disease. The first serious attack of pain about her heart was two years ago at a hotel, when she fainted. During the past three months she had had eight or ten attacks. They usually came on when she was worried or very anxious. She got cold; the pains began just under the left breast and shot into the arm and up the neck. During the attacks her physician said she had often been quite hysterical, tossing herself about, and talking in a very incoherent way. The apex beat of the heart was a little outside the normal position; the aortic second sound was intensified, and at midsternum, opposite the third costal cartilage and along the left border of the sternum, there was a soft diastolic murmur. The pulse was regular, a little jerky; the vessel wall was a little firmer than normal. When

I saw her she was having pretty frequent attacks, and was very nervous and hysterical. After removal from her home surroundings, with rest and quiet, and a course of tonics, she improved very greatly. After my examination I made the following note: "Do the attacks represent true or hysterical angina? That there is a strong neurotic element is undoubted, but the presence of aortic insufficiency, a condition which had been recognized by several physicians some years ago, makes the diagnosis a little dubious." I have seen the patient at intervals during the past six years, and she has had no recurrences of the attacks, and has been in excellent health.

The extreme rarity of true angina in women must always be borne in mind, and also the infrequency of its association, as noted in Lecture II, with mitral-valve disease. Flushes, paræsthesiæ, and various nervous or hysterical manifestations, and particularly the vaso-dilator type of phenomena, suggest strongly pseudo-angina, even though a loud mitral murmur be present. I saw with Dr. Clark, of Kingston, Ontario, a very puzzling case of this kind, the notes of which I have unfortunately mislaid.

In men, while true angina may coexist with apparently normal cardio-vascular condition, in a very large proportion of all cases there are signs of greatly heightened blood pressure, or of sclerosis of the arteries, with a ringing metallic aortic second sound; sometimes only the signs of a weak heart. In men under the age of forty the existence of syphilis should be suspected, as a by no means inconsiderable number of cases result from an aortitis, causing great swelling of the intima and narrowing of the orifices of the coronaries.

In men, too, the question of tobacco has always to be considered, as recurring paroxysms of really maximum intensity may be due to persistent smoking or chewing.

In determining between a true and a false angina, the phenomena of the attack offer most valuable differential cri-

teria. The character of the pain in pseudo-angina, while it may be very severe, rarely has the agonizing quality of true angina, and is seldom, if ever, associated with the sensation of impending death. Patients more often complain of a sense of fullness and distention in the heart. The seat of the pain may be in both identical, and I do not think that much stress can be laid on the point that in pseudo-angina the maximum pain is more over the heart and toward its apex, while in the organic disease the chief seat of the pain is toward the base of the heart and over the aorta. You must remember that in true angina the seat of the pain may be entirely away from the chest, and may be, as in Lord Clarendon's father, at the inner aspect of the arm, or about the wrist, or in rare instances confined to the side of the neck, or even to one testis. While in both forms the pain may radiate to the side of the neck and to the left arm, in pseudo-angina the associated nervous sensations are apt to be much more widespread. There may be numbness and tingling in both extremities, or prior to the onset there may be a feeling of pins and needles in the hands and feet. The vaso-motor phenomena are apt to be much more pronounced in pseudo-angina. The attack may be preceded by flushes, by a sensation of great oppression in the back of the neck or head, and then before the onset of the cardiac pain, not with it or following it, there is coldness of the extremities and sometimes a pronounced tremulousness amounting to what is popularly called a nervous chill. Sweating, combined with the pallor, is not so common in pseudo-angina. The paroxysms of false angina rarely have great abruptness of onset, but are preceded by various nervous and hysterical features.

The attitude in angina pectoris vera is one of its most characteristic features. The patient rarely can stir from the spot in which he is attacked. In the hysterical and neuras-

thenic angina there is often great jactitation, seldom immobility. The patient tosses about on the bed with noisy exclamations of pain, or may walk the floor screaming loudly.

The duration of the attack in pseudo-angina is usually more prolonged, lasting perhaps an hour or more. As I have already mentioned, it occasionally happens that even in the true angina, as in Mr. Sumner's case, the patient is able to walk about, finding relief in moderate exercise during the attack.

And, lastly, among the important points of diagnosis are the circumstances which promote the attacks. In true angina the patient can nearly always fix upon some provocation, as muscular effort, mental irritation, an attack of indigestion; whereas in pseudo-angina the attacks are much more apt to occur spontaneously, and rarely are excited by effort.

It must be acknowledged that the diagnosis is not always so easy as you might supose from any glib summary of differential signs; thus just the other day I was consulted by a practitioner from one of the large Western cities, in whose case the existence of certain well-pronounced coincident nervous phenomena seemed alone to clinch the question of diagnosis. The patient was aged about fifty, a strong man, of strong stock, who had been engaged in large general practice for more than twenty-five years. He had never had syphilis, and had been temperate in the use of alcohol and tobacco; somewhat intemperate in coffee. He had lived a life of a good deal of tension, but had been very well, with the exception of at intervals rheumatic and neuralgic pains. He had never had gout. Two years ago, after a long and tiring drive, he went out one evening to make a visit, and while at the patient's door had a very severe attack of pain about the apex of the heart, which lasted for a minute or two, and which frightened him very much. The next day at dinner he was seized

again, but the pain was scarcely so intense; it was duller and more boring in character. He suffered all night, and in the morning had to take a hypodermic injection of morphine. He had no faintness, the circulation was not involved, and there was no sense of impending dissolution. He felt very weak and used up for nearly a week. He had no return whatever of the pain until last October. He had been working very hard, and had lost a great deal of rest. Then he had the pains at intervals, while he was driving, at the table, when walking, or in bed. They were never very severe, and did not interfere with his work. They were chiefly about the apex of the heart, not beneath the sternum. They radiated down the arms, particularly the left, but he has had pains in both arms as far as the wrists, with numbness, and on several occasions he has had pain and numbness in the left arm without the pain about the heart. These attacks persisted on and off all through the winter, until about two months ago. He then had an attack of influenza with fever, and since then he has had a great deal of nervous palpitation of the heart, particularly with emotion, or if his stomach is full. He does not appear ever to have had a severe agonizing attack with sweating and a sense of impending dissolution.

Certainly in a man of over fifty, though his heart was normal, and his arteries not specially sclerotic, and the pulse tension very little raised, such attacks were, to say the least, suggestive of true angina. But on going into his case more fully two circumstances developed, which were, I think, of much moment, indicating probably that he was of a more neurotic temperament than he was willing to confess. Between three and four years ago, when overworked and worried, he had extraordinary attacks of slight spasm of the glottis, which would come on while he was taking food, or at any time if he was very excited. It would be relieved with a

deep, noisy inspiration almost like childcrowing. These attacks passed away, and he has not had them since. But last summer his wife says that he had the most extraordinary attacks of spasm of the gullet, recurring at every meal for nearly six weeks. At the first attempt at swallowing, either of liquids or of solids, there would be a sudden interruption, which he describes as a sort of spasm of the gullet, and he had to wait several minutes for it to pass off before he could take another mouthful. This patient was very nervous and apprehensive that he had true angina, and yet I think the existence of well-marked œsophagismus and of laryngeal spasm three or four years ago are circumstances which suggest a diagnosis of pseudo-angina.

Prognosis.—One of the most distinguishing features of true angina is a consciousness on the part of the patient, in his anguish of mind, that the very citadel of life has been approached. In a severe, long-continued paroxysm all desire of recovery may be absent in the dread lest he should have again to endure the agony. Subjects of the disease may truly be said to stand in jeopardy every hour, yet it is astonishing with what equanimity the affliction is endured. Charles Sumner said: "This *treacherous* disease produces in my mind a positive uncertainty when I go out of my house whether I shall ever enter it again a living man." The duration of the disorder is most uncertain; there are notable cases, such as John Hunter, in which the attacks have recurred at intervals for twenty or more years.

Recovery is quite possible, and there are instances in which the attacks disappear entirely. In June of last year, in consultation out of town about a case of heart disease, Dr. —— mentioned to me his own case as one of exceptional interest. He was a man of fifty years of age, and had been in very active practice. Twenty years ago, he had been for nearly a year

a terrible sufferer with angina. He was under the care of the late Dr. Donaldson, who regarded the attacks as genuine, as there was also well-marked aortic insufficiency. The patient has remained perfectly free for twenty years.

I saw in November, 1886, with Dr. J. William White, a naval officer, aged forty-six years, who had had severe attacks of angina associated with immobility and a sense of great *angor*. He was a powerfully built man, who had lived well and had taken a great deal of heavy exercise. The pulse tension was increased and the aortic second was accentuated. He had been a heavy smoker, but had not had syphilis. He had kept a very accurate account of the attacks, and he had between October 11, 1886, and August 11, 1887, two hundred and thirty-nine, most of them slight, but some of terrible severity. From the date mentioned to the present he has remained perfectly well, and attributes his recovery largely to the use of the iodide of potassium. He stopped smoking at the time of the paroxysms, but has resumed it since without any detriment.

In a disease so notoriously uncertain as true angina, the prognosis must necessarily be most guarded. Fortunately, as I have already said, the character of the attack is such that the patient is very well aware of the extreme hazard of his state. Of the important elements in prognosis, the following are to be considered:

The frequency and severity of the attacks. Recurrence at short intervals of paroxysms of great severity, induced by slight exertion, is of ill omen, particularly if with them there are marked cardiac arrhythmia and signs of dilatation.

The existence of valvular disease does not in itself materially aggravate the prognosis. A large majority of the worst cases of angina show no signs of valvular lesions. The existence of aortic disease renders the patient, of course, much

more liable to myocardial changes and dilatation and the other consequences of progressive failure of the muscular power. The following is a remarkable instance of good health, even vigor, for years with aortic-valve disease and angina of ten years' duration:

Mr. X., aged fifty-one years, Holbrook, Maryland, consulted me June 10, 1895. His general health had always been good. So long as he could remember he had had heart trouble; he had been short of breath on exertion, and had been conscious, as he expressed it, of a sort of grinding in his chest. When a child he had attacks of extensive blotches on the skin with gastro-intestinal pain (Henoch's purpura). He had rheumatism when twenty-two, but no swelling or redness of the joints. With care he has been able to get about and has lived very comfortably, though he has never been able to do heavy work.

He looked pale, a little thin, and had a suggestive cardiac *facies;* there were no signs of any swelling of the joints. There was an inguinal hernia on the right side. The apex beat was in the fifth and sixth spaces just outside the nipple line. There was a large area of cardiac impulse. There was a loud systolic murmur at the apex region, propagated to axilla and loudly along pectoral border. It increased in intensity toward the left margin of sternum. In the whole apex region there was almost silence in diastole, perhaps a faint rumble, and at the apex there was a slight systolic shock. The systolic murmur became very loud over the sternum, and attained a maximum intensity at the second right costal cartilage, where it was rough, harsh, vibratory, and was propagated with great intensity into the vessels. Along the left margin of sternum and as low as the ensiform cartilage there was an extremely soft diastolic murmur. There was no pulsation to be felt in the sternal notch. The pulsation in the superficial arteries was visible; the vessels were a little stiff. The pulse was 100, not collapsing, of medium volume, and gave one the impression of effort.

He had his first attack of angina ten years ago, coming from Chicago. Two years subsequently he had another attack, and had to have morphine. He has had six or seven attacks alto-

gether, the last one seven days ago as he was getting out of bed. On each occasion there has been a single attack; morphine alone controls them.

Ten years ago he evidently had an attack of cardiac breakdown, with great shortness of breath. Subsequently, for three years, he took ten drops of the tincture of digitalis three times a day, without missing, he thinks, a single dose. During the attack he feels very badly; there is immobility and agonizing pain in the chest, he feels as if he was going to die, and he sweats profusely.

A word or two upon an ethical problem which is often very perplexing—viz., What is your duty in the matter of telling a patient that he is probably the subject of an incurable disease? I can give you no hard-and-fast rule; the temperament of the individual himself, his associations and responsibilities, your own convictions as to the seriousness of the condition—all these must be carefully weighed. The question is somewhat theoretical, since in reality the necessity does not often arise. The announcement has already been made, for no man suffers the anguish of a severe paroxysm of angina without a consciousness of the nearness of the Angel of Death. We are sometimes, I confess, placed in positions of the utmost delicacy, since a man may have not the slightest intimation of his parlous state, and you may become aware of the urgent necessity that he should make proper arrangements to protect his wife and children. In such a case a quiet hint as to the uncertainty of the outlook in heart and artery disease may be enough to set him a-thinking; or, in the case of an "even-balanced soul," the whole question may be discussed frankly. One thing is certain: it is not for you to don the black cap, and, assuming the judicial function, take hope from any patient—"hope that comes to all"—and you may dwell with advantage on the aspects of John Hunter's case rather than on those of Thomas Arnold.

Treatment.—"The first and great object of the practitioner on being called on to treat a case of angina will be to make himself acquainted with its individual character. Beginning with the early history of the disease, he will trace it to its present stage, and will endeavor, from the narrative of the patient and from the observation of the whole phenomena presented to him, to form a clear judgment respecting the local condition of the organs in which the characteristic symptoms have their site, and the state of all the other parts of the system which can in any way influence these; in other words, he must endeavor to ascertain the species or variety of angina, according to the distinction formerly pointed out." This clear statement of Sir John Forbes forms a fitting introduction to the discussion of this part of our subject. Successful treatment depends often upon correct diagnosis; but there are cases of angina pectoris brought to the consultant in which diagnosis and prognosis in themselves constitute the treatment. To a man who has felt that judgment has been given against him, the doom pronounced, and whose mind is haunted with the dread of sudden death, the assurance that the condition is functional and curable comes as a reprieve, and may be the one thing necessary to effect the cure.

True Angina.—Determine in the first place, if possible, the existence of any constitutional disease, as syphilis, gout, or diabetes, and the presence or absence of valvular lesions.

(*a*) *General Management.*—Inquire carefully about the exciting causes of the attacks, which differ in different cases. Usually the patient has learned by bitter experience his limitations in certain directions, and knows much better than you can tell him just what to avoid; but you can emphasize the importance of mental worry, exercise, and diet, the three chief factors. Quiet of mind, avoidance of worries and cares, the cultivation of a calm equanimity—with these, or such like

phrases, we try to impress a poor victim who to previous anxieties has now the added burden of a disease the terrible character of which he can appreciate but can not understand. Our words often seem a mockery, and yet they may be helpful in persuading a man to cast off all unnecessary business and to live a life in which there shall be a minimum of friction. Time, too, with its soothing deception, comes to allay the access of early apprehension, and as succeeding attacks pass there may be less and less mental distress. An important question arises here, Shall a man with angina give up his business? In a majority of cases this sacrifice is unnecessary; the literature abounds with examples of men who, like John Hunter, have done the best work of their lives after the onset of angina. There is so much uncertainty that no rule can be laid down; each individual case must be considered separately. The patient's age, occupation, and, above all, the condition of the vascular system, must be taken into account. Even after a most severe attack, followed by a cardiac breakdown of several months' duration, a man may be able to resume work, and, as in Case V, referred to in Lecture III, be benefited by the steady occupation.

Exercise must be taken within the limits which each individual soon learns to recognize. In severe recurring attacks induced by slight muscular efforts, a period of absolute rest should be enjoined. The sudden, quick movements which rapidly increase the blood pressure and throw a strain upon the heart are the most dangerous; and most of all those with which are associated strong emotions. The patient should be urged to walk on the level, in the literal as well as metaphorical meaning of the phrase. He should learn "to live within the income of his circulation," with which wise saw from the lips of the late Dr. Sibson a friend with organic heart disease has been comforted and sustained for a quarter

of a century. Steady, quiet exercise should be encouraged, except, of course, when there are special signs of cardiac weakness, in which case the resistance gymnastics of the Schott method may be tried.

Diet is in many cases the central point in the treatment. The subjects of angina are often men with large appetites, accustomed to eat freely of rich and strong foods. First, limit the amount taken, which in most persons above forty years of age is far too great; second, see that the quality is suitable by excluding from the dietary rich, highly seasoned foods and those which favor fermentation; and third, arrange the hours for eating. The subjects of angina are usually aware of the necessity of limiting the quantity of food and drink taken at one time. So soon as the stomach is distended there may be warnings of distress about the heart, or in aggravated cases a full meal may always cause an attack. As one patient expressed it, "Had I not to eat, I would never suffer." Light meals should be the rule in all cases; at breakfast and at midday dinner more may be taken than at the evening meal. Late suppers should be interdicted—there is "death in the pot" for angina victims, and a surfeit may be as fatal as poison.

The quality of the food is equally important. Special dietaries may be necessary for patients with gout and glycosuria, but in ordinary cases the food is to be regulated with reference to one all-important feature—viz., flatulence. As you may remember, almost every one of the old writers laid the greatest stress upon this element in the causation of the attacks, and they were right. In dealing with the question of diet we are too apt to adopt some fad to which, with Procrustean precision, we fit every case. A more rational way is to recognize the extraordinary peptic diversity in our patients —in no respect more strikingly shown than in this very mat-

ter of flatulency. Beyond the generally accepted restriction of the carbohydrates we can not go very far without meeting individual peculiarities which have to be considered. The patient himself has to be consulted carefully. Some of you may call to mind what our distinguished colleague Dr. Smollett makes one of his characters, Matt. Bramble, say in *Humphrey Clinker:* "For my own part, I have had a hospital these fourteen years within myself, and studied my own case with the most painful attention, consequently may be supposed to know something of the matter." We are too apt to forget this. An intelligent man should be able to tell you just what articles of food cause most disturbance and produce wind in the stomach or bowels. The fault may not lie in the food, but in the inability of the stomach and bowels to digest it properly. The obese, flabby subjects of angina—not the most numerous class in my experience—and those with weak heart and arterio-sclerosis are specially prone to flatulence. A few doses of blue mass, an occasional saline purge, and the use at times of a good bitter tonic keep this condition in check. The use of hot water before meals, particularly before breakfast, has been found very serviceable.

In elderly men accustomed to stimulants, hot grog at bedtime allays the tendency to flatulency, which is sometimes the cause of wakefulness, or which is apt to disturb the patient in the early morning hours. Peppermint, spirits of camphor, Hoffmann's anodyne, carbolic acid, iodine, and creosote are useful for "wind on the stomach." For the intestinal flatulency a saline purge is often a good corrective; the various supposed intestinal disinfectants may be tried—salol, beta-naphthol, and corrosive sublimate, of which pilules of from one sixtieth to one thirtieth of a grain may be given sometimes with advantage.

(*b*) *General Medical Treatment.*—Of constitutional con-

ditions underlying angina pectoris and capable of treatment, syphilis and gout are the most important. Genuine angina in a man under thirty-five years of age should arouse a suspicion of syphilis, and vigorous measures should be adopted. In gouty cases free elimination by the bowels, skin, and kidneys should be secured, a proper diet ordered, and at intervals a course of colchicum may be prescribed.

One patient, Dr. ——, emphasized repeatedly the benefit he had derived from colchicum. Stimulants should be avoided. Glycosuria is usually controlled by diet, and rarely gives much trouble.

In a large proportion of all cases of angina pectoris the treatment consists in the administration of the iodides and nitrites, remedies which are believed to influence arterial function and arterial nutrition. The use of the iodides of potassium and sodium in this disorder has been advocated most warmly by Huchard, who states that of eighty patients with organic angina treated thoroughly by these drugs twenty-two recovered, forty-three were greatly benefited, and fifteen died.* The iodides appear to have a beneficial effect in checking or modifying the progress of arterio-sclerosis and in lowering the blood pressure. They may influence, too, arterial pain. I have called your attention repeatedly to the influence of iodide of potassium in aneurysm of the aorta, in which the relief of the pain is one of its most striking effects. While I can not say that my experience is in every way so favorable as Huchard's, I can testify to the great relief which has followed its use in many cases, and in a few an apparent cure. Cases which were thoroughly treated nearly ten years ago remain quite well, and I have had within the past three years several patients who have been greatly benefited. I usually order

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* *Le Traitement de l'angine de poitrine*, Paris, 1892.

the iodide of potassium in doses of ten or fifteen grains three times a day. Should it disagree, which is very seldom, I give the sodium salt. Larger doses are not often necessary. If intolerance develops, stop the use for a week and begin with smaller doses. The success in treatment depends upon the perseverance with which the drug is used. On this point let me quote from Huchard's pamphlet: "One of the principal conditions of success is perseverance—constancy in the medication. The drug must be taken for a period of two to four years, in daily doses of one to three grammes, until all symptoms of angina have disappeared for many months, and I hold that a permanent and definitive recovery is not obtainable except after many years of treatment." Reasonable caution must be employed, and you would not give the iodides in patients with advanced arterial degeneration, a dilated heart, and signs of interstitial nephritis. The patients who stand the treatment well are the robust, middle-aged men in whom the angina is the sole symptom. With aortic disease, if fairly compensated, the drug may be used.

The nitrites in hypertension and angina pectoris are of value quite equal to the iodides. The nitrite of amyl is employed in the paroxysm. The nitroglycerin or trinitrin is indicated in all cases in which the tension is persistently high. Given properly, it is a very valuable remedy, but to get any advantage from its use each case must be taken by itself. In the first place, be sure that the nitroglycerin, either in solution or tablets, is fresh. The tablets containing one one-hundredth of a grain are, as a rule, reliable. It is well to begin with only one of these three times a day. The dose may be increased gradually until the patient takes four or five three times a day, or even a larger dose. If the patient notices a slight glow or flush and a little sensation of fullness in the head you may know that the remedy is acting. I feel sure

that in individual cases we often do not employ the drug in sufficient doses. I have never seen it do any harm. The extreme flushing and throbbing headache give reliable indications when the limit has been reached. I have given as much as thirty minims of the one-per-cent. solution, three times a day, to a case of chronic arterio-sclerosis, without any disturbance. The nitrite of sodium, recommended by Hay, may also be tried in doses of five to ten grains three times a day.

Among other remedies which are useful in the general medical treatment of angina, arsenic is sometimes very valuable. Balfour advises it particularly in the weak heart of elderly people, when associated with pain of any kind. In cases of feeble heart with anæmia, iron and strychnine are most valuable remedies, and in order not to trouble the patient with too many doses, arsenic, iron, and strychnine may be given together in compressed powder or pills.

(*c*) *Treatment of the Attack.*—So frequently is the paroxysm excited by gastro-intestinal disturbances that the subjects of angina should not only be warned to be on their guard in this matter, but should be prepared to take prompt measures on the first indication of any distress. No doubt it was from this standpoint that W. W. Ord made the somewhat paradoxical remark that if restricted to the use of one drug in angina he would prefer sulphate of magnesium to nitrite of amyl. A patient should be told to use a saline purge or blue mass or small doses of calomel when he feels gastro-intestinal uneasiness. It frequently happens that much more prompt treatment is necessary for a condition of flatulency. He should be provided with Hoffmann's anodyne and spirits of camphor; a teaspoonful of each in some peppermint water or hot whisky makes an excellent carminative draught. The combination of morphine, cannabis indica, hyoscyamus, capsicum, peppermint, and spirits of chloroform which is now pre-

pared either in liquid or tablet form as chlorodyne is sometimes very advantageous. In tablet form it is particularly convenient, as it may be carried in the waistcoat pocket.

For the paroxysm itself there are three remedies:

Nitrite of amyl, two to five minims, inhaled from a handkerchief, or from cotton wool placed at the bottom of a wineglass, gives prompt relief in certain cases. The patients are in the habit of carrying the remedy in *perles* containing three to five minims, which can be rapidly broken in a handkerchief and inhaled so soon as the very earliest symptoms of the attack are noticed. The introduction of this drug, in the treatment of angina, by Dr. Lauder Brunton has certainly been a great boon to many sufferers, but too much must not be expected of it. It is singularly uncertain. While in one case the attacks are promptly cut short and almost immediate relief obtained, in others it seems quite inert. Curiously enough, considering that its physiological effect is in dilating the peripheral vessels and relieving the widespread angeiospasm, in my experience it has been less efficacious in the vaso-motor type of the disease than in cases of organic angina. It may produce its effect with great rapidity, as shown by the flushed face of the patient and the increased volume and softness of the pulse, without relieving the pain. It sometimes acts better, given by the mouth, combined with the tincture of capsicum in peppermint water.

Morphine hypodermically is the most useful drug in the attack, and if the pain is not relieved quickly by the nitrite of amyl an injection of a quarter of a grain should be given, and repeated in a half or three quarters of an hour if the patient is not relieved. In one case the nitrite of amyl failed repeatedly to give the slightest relief, but from a quarter to a third of a grain of morphine, hypodermically, never failed to allay the terrible distress, and seemed also to steady and im-

prove the heart's action. A point about the use of morphine in angina which I have never seen mentioned except in the paper by Dr. Burney Yeo in the *Practitioner*, already referred to, is the remarkable tolerance of morphine in certain cases. In reporting Case XXXII I mentioned that this patient received between ten o'clock on Saturday night and 1 P. M. on Sunday five grains of morphine hypodermically and by the mouth, which relieved the pain but did not give him sleep. There are cases in which a hypodermic injection of a quarter of a grain of morphine given at the first indication of the attack, as a numbness in the hand or tingling in the fingers, checks it at once.

And third, in any paroxysm of great intensity, while waiting for the nitrite of amyl or morphine to take effect, chloroform may be dropped upon a handkerchief and inhaled. Balfour recommends that it be poured on a sponge in a smelling bottle, and the patient told to breathe it through the nose as deeply as possible. In a minute or two relief is obtained, and as the patient comes under the influence of the drug the bottle drops from his hand, and there is in this way no danger of an overdose. The chloroform acts much more promptly and is much pleasanter to take than ether, and I have never seen any dangerous effects from its use, even in persons with very weak heart's action.

(*d*) *Treatment of the Complications.*—For the syncope of serious attacks the aromatic spirits of ammonia with Hoffmann's anodyne and brandy may be given, or hypodermic injections of ether or camphor. For the dilatation of the heart and cardiac weakness, which sometimes follow the attack, the nitroglycerin with strong frictions to the limbs may favor the circulation at the periphery, while digitalis or digitalin may be given freely to stimulate the heart's action. Digitalin sometimes acts well, as in Case XXXVIII, and may

be given hypodermically. No hard-and-fast rule can be laid down regarding the use of digitalis. It sometimes acts badly, as in a case very carefully studied by W. T. Sharpless, of West Chester. Caffeine and camphor may also be employed. If all these measures seem futile, I would not hesitate to employ puncture of the heart—cardiocentesis—which may arouse to quite vigorous action a dilated and paretic organ. I do not know that this has been employed in the cardiac asystole following a severe paroxysm of angina, but there are instances on record, notably the case of Sloane (*Edinburgh Medical Journal*, vol. xl), in which puncture of the heart with a needle driven firmly into the ventricle has aroused the flagging action apparently without doing the slightest injury.

For the condition of chronic *état de mal angineux*, in which, for a period of many days or even weeks, the patient has recurring attacks with cardiac asthma and feebleness of the circulation, your resources will be taxed to the uttermost. For the dyspnœa and the Cheyne-Stokes breathing full doses of strychnine, hypodermically, may be employed, from a fortieth to a twentieth of a grain, three or four times a day. Special care should be taken that the bowels are kept freely opened. The cardiac measures already spoken of may be employed, and flying blisters to the præcordia and to the bases of the lungs may sometimes give relief.

Treatment of Pseudo-angina Pectoris.—The measures must usually be directed to combating the underlying condition of neurasthenia or hysteria. Occasionally it happens, particularly in medical men, that the mental relief afforded by a positive diagnosis of pseudo-angina is in itself sufficient to effect a cure. Cases II and III, given in Lecture V, are good illustrations of the improvement and permanent cure, up to the present date, of attacks of maximum severity. It is not easy to say to what the rapid relief could be attributed, as the

patients were given only general tonics. In other cases the attacks recur for years, as in the wife of the physician from the Province of Quebec, of whom I spoke, who had had attacks for twenty-five or thirty years. In the severe form, particularly when associated with much vaso-motor disturbance, the Weir Mitchell treatment may be tried with advantage. The effects of seclusion, systematic massage, and electricity, particularly the static form, are sometimes most satisfactory. Where this is not feasible hydrotherapy should be tried, either a systematic course at some institution, or, if this is not practicable, the systematic use of the wet pack at night, followed by thorough friction, will be found advantageous. Some of these cases, particularly if treated at the patient's home, tax to the uttermost the resources of the physician. The change of air and scene in traveling will often be found of advantage.

Drugs are of uncertain and doubtful benefit. We often have to order the bromides and valerian, and in cases with much cardiac irritability and vaso-motor disturbance the use of nitroglycerin in large doses seems sometimes to aid in equalizing and steadying the circulation. In looking over the notes of my cases of pseudo-angina I notice this hopeful feature, that with but one or two exceptions the patients are at present not only alive and well, but free from attacks.

When the attacks of angina are due to the abuse of tobacco, the patient should give up the habit entirely. I do not think there is much risk, either, in stopping abruptly. Counter-irritation over the heart by means of the Paquelin cautery or blisters, the use of strychnine in full doses, and, if the pulse tension is high, of nitroglycerin, are measures which will be found efficacious.

In the worry and strain of modern life arterial degenera-

tion is not only very common, but develops often at a relatively early age. For this I believe that the high pressure at which men live, and the habit of working the machine to its maximum capacity, are responsible, rather than excesses in eating and drinking, or than any special prevalence of syphilis. Angeio-sclerosis, creeping on slowly but surely, "with no pace perceived," is the Nemesis through which Nature exacts retributive justice for the transgression of her laws—coming to one as an apoplexy, to another as an early Bright's disease, to a third as an aneurysm, and to a fourth as angina pectoris, too often slitting "the thin spun life" in the fifth decade, at the very time when success seems assured. Nowhere do we see such an element of tragic sadness as in many of these cases. A man who has early risen and late taken rest, who has eaten the bread of carefulness, striving for success in commercial, professional, or political life, after twenty-five or thirty years of incessant toil reaches the point where he can say, perhaps with just satisfaction, "Soul, thou hast much goods laid up for many years: take thine ease," all unconscious that the fell sergeant has already issued the warrant. How true to life is Hawthorne in the *House of the Seven Gables!* To Judge Pyncheon, who had experienced a mere dimness of sight and a throbbing at the heart—nothing more—and in whose grasp was the meed for which he had "fought and toiled and climbed and crept"; to him, as he sat in the old oaken chair of his grandfathers, thinking of the crowning success of his life, so near at hand, the avenger came through the arteries.

"With what strife and pains we come into the world we know not, but it is commonly no easy matter to get out of it," Sir Thomas Browne says; and, having regard to the uncertainties of the last stage of all, the average man will be of Cæsar's opinion, who, when questioned at his last dinner party

as to the most preferable mode of death, replied—"That which is the most sudden." Against this, one in a string of grievous calamities, we pray in the Litany, though De Quincy insists that the meaning here is "unprepared." In this sense sudden death is rare in angina pectoris, since the end comes but seldom in the first paroxysm. Terrible as are some of these incidental conditions accompanying coronary artery lesions, there is a sort of kindly compensation, as in no other local disease do we so often see the ideal death—death like birth "a sleep and a forgetting."

APPENDIX.

NOTE A.—ROUGNON'S CLAIM (PAGE 6).

WHILE these lectures were in course of publication in the *New York Medical Journal* I had the following explanatory letter from Professor Gairdner, which it is only due to him to publish:

225 ST. VINCENT STREET, GLASGOW, *Sept. 23, 1896.*

MY DEAR OSLER: I have only to-day had my attention directed to your most interesting lectures on angina pectoris in the *New York Medical Journal,* and while appreciating them very much, I must confine my remarks to one point at present, on which I have no doubt you will desire to have my opinion inasmuch as you have formally indicated your own as differing. (Page 178, column 2, as to the case of M. Charles.)

The cause of the difference, however, is this: I was curious to see the original paper of Rougnon, and when in Paris made a special inquiry after it, in vain, both in the Bibliothèque Nationale, and in the library of the École de Médecine (I think). I afterward engaged M. Lereboullet in the search, and he was kind enough to hunt up for me what he thought to be the only copy accessible after considerable research (I think it was at Besançon, but am not sure). He further was good enough to copy, or get copied, for me all that he thought essential in the paper, and sent it over with the remark that *he* could find nothing like A. P. in it. To me it was just the same, but as I unfortunately mislaid his extract I could not precisely refer to it in writing to the *Lancet.*

You have been more fortunate in finding what I can not help supposing to be a quite different document in your marvelous library at Washington. I can only plead that my remarks applied quite correctly to the extract sent to me, and I should be glad if this were made clear, though I can not now fully explain it.

Have you any idea, in America, as to the proper pronunciation of angina? For years I always pronounced it with the ī long, and never once heard it otherwise till Dr. Houghton, of Dublin, pulled me up. I then made an elaborate inquiry into the classical authorities, and found that it comes out apparently clearly that the ĭ is short, as in the test passage in Plautus's *Trinummus*, which has been annotated, so my colleague Professor Ramsey tells me. Is it worth while to make the change? In haste,

Yours very truly, W. T. Gairdner.

NOTE B.—THE CASE OF MR. MATTHEW ARNOLD (PAGE 25).

Matthew Arnold, the distinguished son of Dr. Thomas Arnold, died suddenly on Sunday afternoon, April 15, 1888, in his sixty-sixth year. The various stages in the progress of his disease are well given in his letters. The first intimation we have of any trouble is in a letter to his son, dated May, 1885: "I have been having a horrid pain across my chest, and on Friday mamma carried me to Andrew Clark, who has put me on the strictest of diets for one week—no medicine, but soup, sweet things, fruit, and, worst of all, all green vegetables entirely forbidden, and my liquors confined to one small half glass of brandy with cold water at dinner. I am to see how this suits me. He thinks the pain is not heart, but indigestion. At present I feel very unlike lawn tennis, as going fast or going uphill gives me the sense of having a mountain on my chest; luckily, in fishing, one goes slow and stands still a great deal."

To his daughter about the same time he writes: "I can not get rid of the ache across my chest when I walk; imagine my having to stop half a dozen times in going up to Pains Hill! What a mortifying change! But so one draws to one's end."

On August 26th he writes to his wife from Wales: "On the

whole, I did more yesterday, and did it easier, than I have done since I was first visited by this pain."

On January 11, 1886, in a letter to his daughter, he writes: "I got on very well, and the skating did not bring on the chest pain; smooth motion does not, but laborious motion—making my way uphill or through snow."

During his second visit to America in 1886 he had a very narrow escape from drowning. "The accident was nothing; a wave carried me heavily against a taut rope under water, put there for the safety of bathers; but the shock exhausted me rather, and was followed by a week or so of troublesome attacks of pain across the chest."

On November 27th of the same year he writes to his daughter: "I am quite my old self again—walked about London all yesterday in the fog without choke and pain."

On December 22d, in a letter to Professor Norton, he writes: "If I go too quick I am stopped by a warning in my chest; but I can go about as much as I like if I go leisurely, and I have no attacks of sharp pain. There were some nights in America when I thought that my 'grand climacteric'—an epoch in life which I used to hear a great deal of from my dear mother—would see the end of me; and I think, by the way you looked at me once or twice at Ashfield, you thought so too."

In a letter to Mrs. Coates, January 29, 1887, he wrote: "One should try to bring one's self to regard death as a quite natural event, and surely in the case of the old it is not difficult to do this. For my part, since I was sixty I have regarded each year, as it ended, as something to the good beyond what I could naturally have expected. This summer in America I began to think that my time was really coming to an end. I had so much pain in my chest, the sign of a malady which had suddenly struck down in middle life, long before they came to my present age, both my father and grandfather."

In a letter to Professor Norton he again refers to the "bad attacks of pain while I was with you, the worst I had in America, the worst I have ever had."

There are no further references, and we know that he went down to Liverpool to meet the steamer Aurania, and on Sun-

day afternoon, April 15, 1888, died suddenly in his sixty-sixth year, about three years after the first manifestations of angina.

NOTE C.—RETENTION OF CONSCIOUSNESS AFTER APPARENT CESSATION OF HEART'S ACTION (PAGE 55).

A very remarkable fact in certain cases of angina is the persistence of consciousness, with the ability to engage in conversation and even to walk, after pulsations have ceased at the wrist, or even after the heart beats can no longer be felt. Dr. Macrae, of Council Bluffs, has sent me notes of the following remarkable instance of the kind. A physician who had been the subject of angina, while waiting for Dr. Macrae in his reception room, was seized with an attack. "When I came into the room he was unconscious, with his head dropped over the back of the chair. He was pulseless; no cardiac sound could be heard. He regained consciousness and, with my assistance, walked into the other room and lay upon the lounge. Careful examination again failed to reveal any cardiac movements. He was not in pain, was sensible, but seemingly dazed. He asked me whether his heart had ceased action. I told him it had. He gave a short loving message to his wife, ejaculated, 'Lord have mercy on me!' became unconscious, and died then in a few seconds. He must have lived at least five minutes after I found him. When laid on the lounge he burst into a most profuse perspiration, and breathing was somewhat labored. The point I wish to make is that he lived, was rational, could almost walk by himself, and talked for several minutes after his heart, so far as could be determined, had ceased to beat." In Case XXIII I was very much impressed by this retention of complete consciousness and capability of engaging in conversation when the pulse at the wrist could not be felt.

THE END.

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